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I
MEDICO-CHIRURGICAL
TRANSACTIONS.

PUBLISHED BY

THE ROYAL
MEDICAL AND CHIRURGICAL SOCIETY
OF
LONDON.

—
VOLUME THE SIXTIETH.
—

LONDON:
LONGMANS, GREEN, READER, AND DYER,
PATERNOSTER ROW.

—
1877.

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SECOND SERIES.
VOLUME THE FORTY-SECOND.



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1877.

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OF LONDON.

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- 1810. SIR HENRY HALFORD, BART., M.D., G.C.H.
- 1813. SIR GILBERT BLANE, BART., M.D.
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- 1819. SIR ASTLEY PASTON COOPER, BART., K.C.H., D.C.L.
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- 1823. JOHN ABERNETHY.
- 1825. GEORGE BIRKBECK, M.D.
- 1827. BENJAMIN TRAVERS.
- 1829. PETER MARK ROGET, M.D.
- 1831. SIR WILLIAM LAWRENCE, BART.
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- 1849. THOMAS ADDISON, M.D.
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- 1855. CÆSAR HENRY HAWKINS.
- 1857. SIR CHARLES LOCOCK, BART., M.D.
- 1859. FREDERIC CARPENTER SKEY.
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- 1877. CHARLES WEST, M.D.

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FOR THE SESSION OF 1877-78.

BARCLAY, ANDREW WHYTE, M.D.
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POLLOCK, JAMES E., M.D.
POWER, HENRY.
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FELLOWS

OF THE

ROYAL MEDICAL AND CHIRURGICAL SOCIETY OF LONDON.

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P.—President.

T.—Treasurer.

L.—Librarian.

V.P.—Vice-President.

S.—Secretary.

C.—Member of Council.

The figures succeeding the words *Trans.* and *Pro.* show the number of Papers which have been contributed to the Transactions or Proceedings by the Fellow to whose name they are annexed. *Sci. Com.* is attached to the names of those who have served on the Scientific Committees of the Society.

OCTOBER, 1877.

Those marked thus (†) have paid the Composition Fee in lieu of further annual subscriptions.

Amongst the non-residents those marked thus (*) are entitled by composition to receive the Transactions.

Elected

- 1846 *ABERCROMBIE, JOHN, M.D., Physician to the Cheltenham General Hospital ; 13, Suffolk square, Cheltenham.
- 1851 *ACLAND, HENRY WENTWORTH, M.D., F.R.S., Honorary Physician to H.R.H. the Prince of Wales ; Physician to the Radcliffe Infirmary ; Regius Professor of Medicine, and Clinical Professor in the University of Oxford.
- 1847 ACOSTA, ELISHA, M.D., 24, Rue de Luxembourg, St. Honoré, Paris.

Elected

- 1852 ADAMS, WILLIAM, Surgeon to the Great Northern Hospital, and to the National Hospital for Paralysed and Epileptic; Consulting Surgeon to the National Orthopædic Hospital, Great Portland street; 5, Henrietta street, Cavendish square. C. 1873-4. *Trans.* 3.
- 1867 AIKIN, CHARLES ARTHUR, 7, Clifton place, Hyde park.
- 1837 *AINSWORTH, RALPH FAWSETT, M.D., Consulting Physician to the Manchester Royal Infirmary; Cliff Point, Lower Broughton, Manchester.
- 1839 ALCOCK, SIR RUTHERFORD, K.C.B., K.C.T., K.T.S., D.C.L., late H.M.'s Envoy Extraordinary at the Court of Peking. *Trans.* 1.
- 1866 ALLBUTT, THOMAS CLIFFORD, M.A. and M.D., F.L.S., Lecturer on the Practice of Physic at the Leeds School of Medicine, and Physician to the Leeds General Infirmary; 35, Park square, Leeds. *Trans.* 3.
- 1863 ALTHAUS, JULIUS, M.D., Physician to the Infirmary for Epilepsy and Paralysis; 18, Bryanston street, Portman square. *Trans.* 2.
- 1862 ANDREW, EDWYN, M.D., Hardwick House, St. John's Hill, Shrewsbury.
- 1862 ANDREW, JAMES, M.D., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 22, Harley street, Cavendish square. *Trans.* 1.
- 1820 ANDREWS, THOMAS, M.D., Norfolk, Virginia.
- 1870 ARNOTT, HENRY, Chichester.
- 1819 †ARNOTT, JAMES MONCRIEFF, F.R.S., Chapel House, Lady Bank, Fifeshire, and 36, Sussex gardens, Hyde park. L. 1826-8. V.P. 1832-3. T. 1835-40. C. 1846, 1855-6. P. 1847-8. *Trans.* 8.
- 1817 †ASHBURNER, JOHN, M.D., F.L.S., 161A, Piccadilly. C. 1821, 1830-31.
- 1874 AVELING, JAMES H., M.D., Physician to the Chelsea Hospital for Women; 1, Upper Wimpole street, Cavendish square.

Elected

- 1836 BAIRD, ANDREW WOOD, M.D., Physician to the Dover Hospital ; 7, Camden crescent, Dover, Kent.
- 1851 *BAKER, ALFRED, Surgeon to the Birmingham General Hospital ; 20A, Temple row, Birmingham.
- 1873 *BAKER, J. WRIGHT, Senior Surgeon to the Derbyshire General Infirmary ; 102, Friargate, Derby.
- 1865 BAKER, WILLIAM MORRANT, Assistant Surgeon to, and Lecturer on Physiology at, St. Bartholomew's Hospital ; Surgeon to the Evelina Hospital for Sick Children ; 26, Wimpole street, Cavendish square. *Trans.* 4.
- 1869 BAKEWELL, ROBERT HALL, M.D., Dunedin, New Zealand.
- 1839 †BALFOUR, THOMAS GRAHAM, M.D., F.R.S., Surgeon General ; Coombe Lodge, Wimbledon Park. C. 1852-3. V.P. 1860-1. T. 1872. *Trans.* 2.
- 1848 BALLARD, EDWARD, M.D., Inspector, Medical Department, Local Government Board ; 12, Highbury terrace, Islington. C. 1872. V.P. 1875-6. *Trans.* 5.
- 1866 *BANKS, JOHN THOMAS, M.D., Physician to Richmond, Whitworth, and Hardwicke Hospitals ; Consulting Physician to the Coombe Hospital ; 10, Merrion square east, Dublin.
- 1847 BARCLAY, ANDREW WHYTE, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital ; Medical Officer of Health for Chelsea ; 23A, Bruton street, Berkeley square. S. 1857-60. L. 1861-2. C. 1865-6. V.P. 1872-3. *Trans.* 2.
- 1862 BARKER, EDGAR, 21, Hyde park street.
- 1833 †BARKER, THOMAS ALFRED, M.D., Consulting Physician to St. Thomas's Hospital ; 27, Wimpole street. C. 1844-5. V.P. 1853-4. T. 1860-2. *Trans.* 6.
- 1876 BARLOW, THOMAS, M.D. and B.S. Lond., Assistant Physician to Charing Cross Hospital, and to the Hospital for Sick Children ; 10, Montague street, Russell square.

Elected

- 1861 BARNES, ROBERT, M.D., Obstetric Physician to, and Lecturer on Midwifery at, St. George's Hospital; 31, Grosvenor street. C. 1877. *Trans.* 4.
- 1864 BARRATT, JOSEPH GILLMAN, M.D., 8, Cleveland gardens, Bayswater.
- 1840 BARROW, BENJAMIN, Surgeon to the Royal Isle of Wight Infirmary; Southlands, Ryde, Isle of Wight.
- 1859 BARWELL, RICHARD, Surgeon to the Charing Cross Hospital; 32, George street, Hanover square. C. 1876-77. *Trans.* 3.
- 1868 BASTIAN, HENRY CHARLTON, M.A., M.D., F.R.S., Professor of Pathological Anatomy in University College, London, and Physician to University College Hospital; 20, Queen Anne street, Cavendish square. *Trans.* 1.
- 1874 BAXTER, EVAN BUCHANAN, M.D., Assistant Physician to King's College Hospital; Professor of Materia Medica at King's College; 28, Weymouth street, Portland place.
- 1875 BEACH, FLETCHER, M.B., Medical Superintendent, Clapton Idiot Asylum, Lower Clapton.
- 1862 BEALE, LIONEL SMITH, M.B., F.R.S., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street. C. 1876-77. *Trans.* 1.
- 1860 *BEALEY, ADAM, M.D., M.A. Camb., Oak Lea, Harrogate.
- 1856 BEARDSLEY, AMOS, F.L.S., Bay villa, Grange-over-Sands, Lancashire.
- 1871 BECK, MARCUS, M.S., Assistant Surgeon to University College Hospital; 30, Wimpole street, Cavendish square.
- 1858 BEGLEY, WILLIAM CHAPMAN, A.M., M.D., late of the Middlesex County Lunatic Asylum, Hanwell; 26, Saint Peter's square, Hammersmith. C. 1877.
- 1819 †BELL, THOMAS, F.R.S., F.L.S., The Wakes, Selborne, Hants. C. 1832-3. V.P. 1854. *Trans.* 1.

Elected

- 1871 BELLAMY, EDWARD, Senior Assistant-Surgeon to, and Lecturer on Anatomy at, Charing Cross Hospital; Profeseor of Anatomy in the Science and Art Department, South Kensington; 59, Margaret street, Cavendish square.
- 1847 BENNET, JAMES HENRY, M.D., The Ferns, Weybridge, and Mentone.
- 1845 †BERRY, EDWARD UNWIN, 76, Gower street, Bedford square.
- 1820 BERTIN, STEPHEN, Paris.
- 1872 BEVERLEY, MICHAEL, M.D., Assistant Surgeon to the Norfolk and Norwich Hospital; 63, St. Giles's street, Norwich.
- 1865 *BICKERSTETH, EDWARD ROBERT, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Clinical Surgery in the Liverpool Royal Infirmary School of Medicine; 2, Rodney street, Liverpool. *Trans.* 1.
- 1815 †BILLING, ARCHIBALD, M.D., F.R.S., Member of the Senate of the University of London; 34, Park lane. C. 1825. V.P. 1828-9.
- 1854 BIRD, PETER HINCKES, F.L.S., Medical Officer of Health for the Fylde Union, West Lancashire; 4, Clifton terrace, Lytham, Lancashire, and 1, Norfolk square, Sussex gardens, Hyde park.
- 1856 BIRD, WILLIAM, Consulting Surgeon to the West London Hospital; Bute House, Hammersmith.
- 1849 BIRKETT, EDMUND LLOYD, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest; 48, Russell square. C. 1865-6.
- 1851 †BIRKETT, JOHN, F.L.S., *Treasurer*, Consulting Surgeon to Guy's Hospital; Corresponding Member of the "Société de Chirurgie" of Paris; Inspector of Anatomy for the Provinces in England and Wales; 59, Green street, Grosvenor square. L. 1856-7. S. 1863-5. C. 1867-8. T. 1870-76. *Trans.* 8. *Sci. Com.*
- 1866 BISHOP, EDWARD, M.D., Cintra park, Upper Norwood.
- 1843 †BLACK, PATRICK, M.D., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 11, Queen Anne street, Cavendish square, C. 1856. V.P. 1866. T. 1869-70.

Elected

- 1840 BLAKISTON, PEYTON, M.D., F.R.S.
- 1865 BLANCHET, HILARION, Examiner to the College of Physicians and Surgeons, Lower Canada ; 6, Palace street, Quebec, Canada east.
- 1865 BLANDFORD, GEORGE FIELDING, M.D., Lecturer on Psychological Medicine at St. George's Hospital ; 71, Grosvenor street.
- 1867 BLOXAM, JOHN ASTLEY, Assistant-Surgeon to Charing Cross Hospital ; Surgeon for Out-Patients to the Lock Hospital ; Junior Surgeon to the West London Hospital ; 8, George street, Hanover square.
- 1823 BOJANUS, LOUIS HENRY, M.D., Wilna.
- 1846 †BOSTOCK, JOHN ASHTON, C.B., Hon. Surgeon to H.M. the Queen ; Surgeon-Major, Scots Fusilier Guards ; 73, Onslow gardens, Brompton. C. 1861-2. V.P. 1870-71. *Sci. Com.*
- 1869 BOURNE, WALTER, M.D. [care of the National Bank of India, 80, King William street, City.]
- 1870 *BOWLES, ROBERT LEAMON, M.D., 8, West terrace, Folkestone.
- 1841 †BOWMAN, WILLIAM, F.R.S., F.L.S., Surgeon to the Royal London Ophthalmic Hospital, Moorfields ; 5, Clifford street, Bond street. C. 1852-3. V.P. 1862. *Trans.* 3.
- 1862 BRACE, WILLIAM HENRY, M.D., 7, Queen's Gate terrace, Kensington.
- 1874 BRADSHAW, A. F., Surgeon-Major ; Surgeon to the Rt. Hon. the Commander in Chief in India ; Army Head Quarters, Bengal Presidency. [Agent : Vesey W. Holt, 17, Whitehall place.]
- 1867 *BRETT, ALFRED T., M.D., Watford, Herts.
- 1876 BRIDGES, ROBERT, M.B., Consulting Physician to St. Bartholomew's Hospital ; 52, Bedford square.
- 1867 BRIDGEWATER, THOMAS, M.B. Lond., Harrow-on-the-Hill, Middlesex.

Elected

- 1868 BROADBENT, WILLIAM HENRY, M.D., Physician to, and Joint Lecturer on Medicine at, St. Mary's Hospital; Physician to the London Fever Hospital; 34, Seymour street, Portman square. *Trans.* 2.
- 1851 †BRODHURST, BERNARD EDWARD, F.L.S., Surgeon to the Royal Orthopædic Hospital; 20, Grosvenor street. C. 1868-9. *Trans.* 2. *Pro.* 1.
- 1872 BRODIE, GEORGE BERNARD, M.D., Consulting Physician-Accoucheur to Queen Charlotte's Hospital; 56, Curzon street, Mayfair. *Trans.* 1.
- 1844 †BROOKE, CHARLES, M.A., F.R.S., Consulting Surgeon to the Westminster Hospital; 16, Fitzroy square. C. 1855. L. 1866-72. V.P. 1875-76.
- 1860 BROWN-SÉQUARD, CHARLES EDOUARD, M.D., F.R.S., Laureate of the Academy of Sciences of Paris; New York, United States. *Sci. Com.*
- 1874 BRUCE, JOHN MITCHELL, M.D., Assistant Physician to, and Lecturer on Materia Medica at, the Charing Cross Hospital; Assistant Physician to the Hospital for Consumption, Brompton; 60, Queen Anne street.
- 1867 BRUNJES, MARTIN, 27, Edgware road.
- 1871 BRUNTON, THOMAS LAUDER, M.D., F.R.S., Assistant Physician to, and Lecturer on Materia Medica at, St. Bartholomew's Hospital; Examiner in Materia Medica at the University of London; 50, Welbeck street, Cavendish square.
- 1860 BRYANT, THOMAS, Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 53, Upper Brook street, Grosvenor square. C. 1873-4. *Trans.* 8; *Pro.* 1. *Sci. Com.*
- 1855 BRYANT, WALTER JOHN, M.R.C.P. Edinb.; Physician to the Home for Incurable Children, Maida vale; 23A, Sussex square, Hyde park gardens.
- 1823 BUCHANAN, B. BARTLET, M.D.
- 1864 BUCHANAN, GEORGE, M.D., Inspector, Medical Department, Local Government Board; 24, Nottingham place, Marylebone road.

Elected

- 1864 BUCKLE, FLEETWOOD, M.D.
- 1876 BUCKNILL, JOHN CHARLES, M.D. ; 39, Wimpole street.
- 1839 BUDD, GEORGE, M.D., F.R.S., Consulting Physician to the Seamen's Hospital, Greenwich ; Ashleigh, Barnstaple. C. 1846-7. V.P. 1857. *Trans.* 5.
- 1833 †BURROWS, SIR GEORGE, Bart., M.D., D.C.L., F.R.S., Physician in Ordinary to H.M. the Queen ; Consulting Physician to St. Bartholomew's Hospital ; Member of the Senate of the University of London ; 18, Cavendish square. C. 1839-40, 1858-9. T. 1845-7. V. P. 1849-50. P. 1869-70. *Trans.* 2.
- 1837 †BUSK, GEORGE, F.R.S., F.L.S., Consulting Surgeon to the Seamen's Hospital, Greenwich ; Member of the Senate of the University of London ; 32, Harley street, Cavendish square. C. 1847-8. V.P. 1855. T. 1866. *Trans.* 4.
- 1873 BUTLIN, HENRY TRENTAM, Surgical Registrar to St. Bartholomew's Hospital ; Assistant Surgeon to the West London Hospital ; 47, Queen Anne street, Cavendish square. *Trans.* 2.
- 1871 BUTT, WILLIAM F., 25, Park street, Park lane.
- 1868 BUZZARD, THOMAS, M.D., Physician to the National Hospital for the Paralysed and Epileptic ; 56, Grosvenor street, Grosvenor square.
- 1851 *CADGE, WILLIAM, Surgeon to the Norfolk and Norwich Hospital ; 24, St. Giles's street, Norwich. *Trans.* 1.
- 1861 CALLENDER, GEORGE WILLIAM, F.R.S., Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital ; Surgeon to the Charter House ; Examiner in Anatomy at the University of London ; 7, Queen Anne street, Cavendish square. C. 1874. *Trans.* 4. *Sci. Com.*
- 1875 CARTER, CHARLES HENRY, M.D., Physician to the Hospital for Women, Soho square ; 45, Great Cumberland place.

Elected

- 1853 CARTER, ROBERT BRUDENELL, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. George's Hospital; 69, Wimpole street, Cavendish square, W. *Trans.* 1.
- 1845 †CARTWRIGHT, SAMUEL, late Professor of Dental Surgery at King's College, London, and Surgeon-Dentist to King's College Hospital; Consulting Surgeon to the Dental Hospital; 32, Old Burlington street. C. 1860-1. *Sci. Com.*
- 1868 CAVAFY, JOHN, M.D., Assistant-Physician to, and Lecturer on the Principles and Practice of Medicine at, St. George's Hospital; Physician to the Victoria Hospital for Children; 2, Upper Berkeley street, Portman square.
- 1871 CAYLEY, WILLIAM, M.D., Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital; Physician to the London Fever Hospital and to the North-Eastern Hospital for Children; 58, Welbeck street, Cavendish square.
- 1845 †CHALK, WILLIAM OLIVER, 3, Nottingham terrace, York gate, Regent's park. C. 1872-3.
- 1844 †CHAMBERS, THOMAS KING, M.D., Hon. Physician to H.R.H. the Prince of Wales; Consulting Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Consulting Physician to the Lock Hospital; 24, Mount street, Grosvenor square. C. 1861. V.P. 1867.. L. 1869-72. *Trans.* 1.
- 1859 CHANCE, FRANK, M.D., Burleigh House, Sydenham Hill.
- 1849 CHAPMAN, FREDERICK, Old Friars, Richmond Green, Surrey.
- 1877 CHARLES, T. CRANSTOUN, M.D., Lecturer on Practical Physiology at St. Thomas's Hospital; 10, Mitre court Chambers, Temple.
- 1868 CHEADLE, WALTER BUTLER, M.D., Assistant-Physician to, and Lecturer on Pathology at, St. Mary's Hospital; Assistant-Physician to the Hospital for Sick Children; 2, Hyde park place, Cumberland gate.
- 1873 *CHISHOLM, EDWIN, M.D., Camden, near Sydney, New South Wales.

Elected

- 1865 CHOLMELEY, WILLIAM, M.D., Physician to the Great Northern Hospital, and to the Margaret Street Infirmary for Consumption; 63, Grosvenor street, Grosvenor square.
- 1872 CHRISTIE, THOMAS BEITH, M.D., Medical Superintendent, Royal India Asylum, Ealing.
- 1866 CHURCH, WILLIAM SELBY, M.D., Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 130, Harley street, Cavendish square.
- 1860 CLARK, ANDREW, M.D., Physician to, and Lecturer on Medicine at, the London Hospital; 16, Cavendish square. C. 1875.
- 1839 †CLARK, FREDERICK LE GROS, F.R.S., Consulting Surgeon to St. Thomas's Hospital; 63, Warrior square, St. Leonards-on-Sea. S. 1847-9. V.P. 1855-6. *Trans.* 5.
- 1848 CLARKE, JOHN, M.D., 42, Hertford street, May Fair. C. 1866.
- 1866 CLARKE, WILLIAM FAIRLIE, M.D., M.A. Oxon., Southborough, Tunbridge Wells. *Trans.* 2.
- 1850 CLARKSON, JOSIAH, New Hall street, Birmingham. *Trans.* 1.
- 1842 †CLAYTON, OSCAR MOORE PASSEY, Extra Surgeon-in-Ordinary to H.R.H. the Prince of Wales, and Surgeon-in-Ordinary to H.R.H. the Duke of Edinburgh; 5, Harley street, Cavendish square. C. 1865.
- 1853 CLOVER, JOSEPH THOMAS, 3, Cavendish place, Cavendish square. C. 1873.
- 1857 COATES, CHARLES, M.D., Physician to the Bath General and Royal United Hospitals; 10, Circus, Bath.
- 1868 COCKLE, JOHN, M.D., F.L.S., Physician to the Royal Free Hospital; 7, Suffolk place, Pall mall. *Trans.* 2.
- 1850 COHEN, DANIEL WHITAKER, M.D., South Bank, North Down lane, Bideford, Devon.
- COLELY, DAVIES, see *Davies-Colley*.
- 1854 COLLINS, FREDERICK, M.D., Wanstead Lodge, Essex.

Elected

- 1865 COOPER, ALFRED, Surgeon to the Lock Hospital ; Assistant Surgeon to St. Mark's Hospital ; Surgeon to the West London Hospital ; 9, Henrietta street, Cavendish square.
- 1843 †COOPER, WILLIAM WHITE, Surgeon-Oculist in Ordinary to H.M. the Queen ; Consulting Ophthalmic Surgeon to St. Mary's Hospital ; 19, Berkeley square. C. 1858-9. V.-P. 1873-4.
- 1868 CORNISH, WILLIAM ROBERT, Surgeon-Major, Madras Army ; Sanitary Commissioner for Madras ; Secretary to the Inspector-General, Indian Medical Department.
- 1860 *CORY, THOMAS CHARLES STEUART, M.D., Surgeon to the Belfast General Dispensary ; 146, Donegall Pass, Belfast.
- 1853 CORY, WILLIAM GILLET, M.D., Hengistbury House, Christchurch, Hampshire.
- 1847 †COTTON, RICHARD PAYNE, M.D., *Vice-President*, Consulting Physician to the Hospital for Consumption, Brompton ; 33, Cavendish square. C. 1863. V.P. 1876-7.
- 1864 COULSON, WALTER JOHN, Surgeon to the Lock Hospital, 17, Harley street, Cavendish square.
- 1860 †COUPER, JOHN, Surgeon to, and Lecturer on Surgery at, the London Hospital ; Assistant-Surgeon to the Royal London Ophthalmic Hospital ; 80, Grosvenor street. C. 1876.
- 1877 COUPLAND, SIDNEY, M.D., Assistant Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital ; 7, Nottingham place.
- 1862 COWELL, GEORGE, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital ; Surgeon to the Royal Westminster Ophthalmic Hospital ; Surgeon to the Victoria Hospital for Children ; 19, George street, Hanover square.
- 1841 CRAWFORD, MERVYN ARCHDALL NOTT, M.D. C. 1853-4.

Elected

- 1868 CRAWFORD, THOMAS, M.D., Deputy Inspector-General of Hospitals (India) ; Umbalah, Punjaub.
- 1873 CREIGHTON, CHARLES, M.B., Anatomical Museum, Cambridge.
- 1869 *CRESSWELL, PEARSON R., Dowlais, Merthyr Tydvil.
- 1874 CRIPPS, WILLIAM HARRISON, Surgeon to the Great Northern Hospital ; Assistant Surgeon to the Royal Free Hospital ; Assistant Demonstrator of Anatomy, St. Bartholomew's Hospital ; 61, Pall Mall.
- 1847 CRITCHETT, GEORGE, Surgeon to the Royal London Ophthalmic Hospital, Moorfields ; Ophthalmic Surgeon to the Middlesex Hospital ; 21, Harley street, Cavendish square. C. 1865. V.P. 1872. *Trans.* 1.
- 1868 CROFT, JOHN, Surgeon to, and Lecturer on Operative Surgery at, St. Thomas's Hospital ; 61, Brook street, Grosvenor square.
- 1862 CROMPTON, SAMUEL, M.D., Physician to the Salford Royal Hospital and Dispensary ; 24, St. Ann's square, Manchester.
- 1837 CROOKES, JOHN FARRAR, 5, Waterloo crescent, Dover.
- 1860 CROSS, RICHARD, M.D., Carlton House, Belmont road, Scarborough.
- 1872 CROSSE, THOMAS WILLIAM, Surgeon to the Norfolk and Norwich Hospital ; 22, St. Giles's street, Norwich.
- 1849 *CROWFOOT, WILLIAM EDWARD, Beccles, Suffolk.
- 1846 CURLING, HENRY, Surgeon to the Margate Royal Sea-Bathing Infirmary, and the Ramsgate Seamen's Infirmary ; Ramsgate, Kent.
- 1837 †CURLING, THOMAS BLIZARD, F.R.S., Consulting-Surgeon to the London Hospital ; 39, Grosvenor street. S. 1845-6. C. 1850. T. 1854-7. V.P. 1859. P. 1871-2. *Trans.* 13. *Pro.* 1. *Sci. Com.*
- 1873 CURNOW, JOHN, M.D., Professor of Anatomy at King's College, London, and Assistant Physician to King's College Hospital ; Examiner in Anatomy at the University of London ; 3, Warwick street, Cockspur street.

Elected

- 1847 CURREY, JOHN EDMUND, M.D., Lismore, County Waterford.
- 1822 CUSACK, CHRISTOPHER JOHN, Chateau d'Eu, France.
- 1852 CUTLER, THOMAS, M.D., Spa, Belgium.
- 1872 DALBY, WILLIAM BARTLETT, M.B., Lecturer on Aural Surgery at St. George's Hospital; 18, Savile row. *Trans.* 1.
- 1836 *DANIEL, JAMES STOCK, Ramsgate, Kent.
- 1848 DAUBENY, HENRY, M.D., San Remo, Italy.
- 1874 DAVIDSON, ALEXANDER, M.D., Physician to the Liverpool Northern Hospital; 49, Rodney street, Liverpool.
- 1853 DAVIES, ROBERT COKER NASH, Rye, Sussex.
- 1852 DAVIES, WILLIAM, M.D., 18, Gay street, Bath.
- 1876 DAVIES-COLLEY, JOHN NEVILLE C., M.C., Assistant-Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; 36, Harley street, Cavendish square. *Trans.* 1.
- 1852 DAVIS, JOHN HALL, M.D., Physician Accoucheur to, and Lecturer on Midwifery at, the Middlesex Hospital; Physician to the Royal Maternity Charity, and Consulting Physician-Accoucheur to the St. Pancras Infirmary; Examiner in Obstetric Medicine at the University of London; 24, Harley street, Cavendish square. C. 1869-70.
- 1867 DAY, WILLIAM HENRY, M.D., Physician to the Samaritan Free Hospital for Women and Children; 10, Manchester square.
- 1846 *DENTON, SAMUEL BEST, M.D., Ivy Lodge, Hornsea, Hull.
- 1859 †DICKINSON, WILLIAM HOWSHIP, M.D., Physician to, and Lecturer on Pathology at St. George's Hospital, and Senior Physician to the Hospital for Sick Children; 9, Chesterfield street, Mayfair. C. 1874-5. *Trans.* 13. *Sci. Com.*

Elected

- 1839 †DIXON, JAMES, Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Consulting Ophthalmic Surgeon to the Asylum for Idiots; Harrow Lands, Dorking. L. 1849-55. V.P. 1857-8. T. 1863-4. C. 1866-7. *Trans.* 4.
- 1862 DOBELL, HORACE B., M.D., Consulting Physician to the Royal Hospital for Diseases of the Chest, City road; 84, Harley street. *Trans.* 2.
- 1845 DODD, JOHN.
- 1877 DORAN, ALBAN HENRY GRIFFITHS, Surgeon to Out-Patients, Samaritan Hospital; Pathological Assistant to the Museum of the Royal College of Surgeons of England; 20, Lower Seymour street, Portman square.
- 1863 DOWN, JOHN LANGDON HAYDON, M.D., Physician to the London Hospital; 39, Welbeck street, Cavendish square. *Trans.* 2.
- 1867 DRAGE, CHARLES, M.D., Hatfield, Herts.
- 1853 DRUITT, ROBERT, F.R.C.P. [8, Strathmore gardens, Kensington Mall.] *Trans.* 2.
- 1865 DRYSDALE, CHARLES ROBERT, M.D., Physician to the Farringdon Dispensary; Assistant-Physician to the Metropolitan Free Hospital; 17, Woburn place, Russell square.
- 1865 DUCKWORTH, DYCE, M.D., Assistant-Physician to, and Lecturer on Skin Diseases at, St. Bartholomew's Hospital; 11, Grafton street, Bond street. *Trans.* 1.
- 1876 DUDLEY, WILLIAM LEWIS, M.D., 117, Cromwell road, South Kensington.
- 1845 DUFF, GEORGE, M.D., High street, Elgin.
- 1874 DUFFIN, ALFRED BAYNARD, M.D., Professor of Pathological Anatomy in King's College, London, and Physician to King's College Hospital; 18, Devonshire street, Portland place.
- 1874 DUKA, THEODORE, M.D., Surgeon-Major, H.M.'s Bengal Army; 38, Montagu square.
- 1871 DUKE, BENJAMIN, 1, Cavendish terrace, Clapham Common.

Elected

- 1871 *DUKES, CLEMENT, M.D. and B.S., Horton crescent, Rugby, Warwickshire.
- 1867 DUKES, M. CHARLES, M.D., Wellesley Villa, Wellesley road, Croydon.
- 1833 †DUNN, ROBERT, 31, Norfolk street, Strand. C. 1845-6. *Trans.* 2.
- 1861 DU PASQUIER, CLAUDIUS FRANCIS, Surgeon-Apothecary to H.M. the Queen, and to the Household of H.R.H. the Prince of Wales; 62, Pall Mall.
- 1863 DURHAM, ARTHUR EDWARD, F.L.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 82, Brook street, Grosvenor square. C. 1876-7. *Trans.* 5. *Sci. Com.*
- 1874 DURHAM, FREDERIC, M.B., Surgical Registrar to Guy's Hospital; 38, Brook street, Grosvenor square.
- 1843 DURRANT, CHRISTOPHER MERCER, M.D., Physician to the East Suffolk and Ipswich Hospital; Ipswich, Suffolk.
- 1872 EAGER, REGINALD, M.D., Northwoods, near Bristol.
- 1836 EARLE, JAMES WILLIAM, late of Norwich.
- 1868 EASTES, GEORGE, M.B., Lond. Surgeon-Accoucheur to the Western General Dispensary; 69, Connaught street, Hyde park square.
- 1824 EDWARDS, GEORGE.
- 1823 EGERTON, CHARLES CHANDLER, Kendall Lodge, Epping.
- 1869 ELAM, CHARLES, M.D., Assistant-Physician to the National Hospital for the Paralysed and Epileptic; 75, Harley street, Cavendish square.
- 1861 *ELLIOT, ROBERT, M.D., Physician to the Fever Hospital and to the Dispensary, Carlisle; Coroner for Carlisle; 35, Lowther street, Carlisle.
- 1848 ELLIS, GEORGE VINER, late Professor of Anatomy in University Colleges, London. C. 1863-4. *Trans.* 2.
- 1868 ELLIS, JAMES, M.D., the Sanatorium, Anaheim, Los Angeles County, California.

Elected

- 1854 *ELLISON, JAMES, M.D., Surgeon-in-Ordinary to the Royal Household, Windsor; 14, High street, Windsor.
- 1842 †ERICHSEN, JOHN ERIC, F.R.S., Surgeon Extraordinary to H.M. the Queen; late Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital; 6, Cavendish place, Cavendish square. C. 1855-6. V.P. 1868. *Trans.* 2.
- 1874 EVANS, GEORGE HENRY, M.D., Assistant-Physician to the Middlesex Hospital; Assistant-Physician to the City of London Hospital for Diseases of the Chest, Victoria park; 29, Devonshire street, Portland place.
- 1845 EVANS, WILLIAM JULIAN, M.D.
- 1877 EWART, WILLIAM, M.B., 33, Somerset street, Portman square.
- 1875 *FAGAN, JOHN, Surgeon to the Belfast Hospital for Sick Children; 11, College square north, Belfast.
- 1864 FAGGE, CHARLES HILTON, M.D., Assistant-Physician to, and Lecturer on Pathology at, Guy's Hospital; 11, St. Thomas's street, Southwark. *Trans.* 6.
- 1869 FAIRBANK, FREDERICK ROYSTON, M.D., 46, Hallgate, Doncaster.
- 1858 FALCONER, RANDLE WILBRAHAM, M.D., Physician to the Bath United Hospital; 22, Bennett street, Bath.
- 1862 FARQUHARSON, ROBERT, M.D., Physician in charge of the Skin Department, and Lecturer on Materia Medica at St. Mary's Hospital; Physician to the Belgrave Hospital for Children; 23, Brook street, Grosvenor square.
- 1844 †FARRE, ARTHUR, M.D., F.R.S., Physician Extraordinary to H.M. the Queen; Physician-Accoucheur to H.R.H. the Princess of Wales; 18, Albert Mansions, Victoria street, Westminster. C. 1857. V.P. 1864. *Sci. Com.*

Elected

- 1872 FAYRER, SIR JOSEPH, K.S.I., M.D., F.R.S. Edin., Honorary Physician to H.M. the Queen, and Physician to H.R.H. the Duke of Edinburgh; Surgeon-Major, Bengal Army; Examining Medical Officer to the Secretary of State for India in Council; President of the Indian Medical Board; 16, Granville place, Portman square.
- 1872 *FENWICK, JOHN C. J., M.D., Physician to the Durham County Hospital; Chilton Hall, Ferry hill, and 16, Old Elvet, Durham.
- 1863 FENWICK, SAMUEL, M.D., Physician, with charge of Out-patients, to, and Lecturer on Medicine at, the London Hospital; 29, Harley street, Cavendish square. *Trans.* 3.
- 1852 *FIELD, ALFRED GEORGE.
- 1849 FINCHAM, GEORGE TUPMAN, M.D., Physician to, and Joint Lecturer on Medicine at, the Westminster Hospital; 13, Belgrave road, Pimlico. C. 1871.
- 1866 FISH, JOHN CROCKETT, B.A., M.B. Camb., Junior Physician to the West London Hospital; 92, Wimpole street, Cavendish square.
- 1860 FITZGERALD, THOMAS GEORGE, Surgeon-Major. [6, Whitehall yard.]
- 1866 FITZPATRICK, THOMAS, M.D., M.A., Dublin; Physician to the Western General Dispensary; 30, Sussex gardens, Hyde park.
- 1842 FLETCHER, THOMAS BELL ELCOCK, M.D., Consulting Physician to the Birmingham General Hospital; 7, Waterloo street, Birmingham. *Trans.* 1.
- 1864 *FOLKER, WILLIAM HENRY, Surgeon to the North Staffordshire Infirmary; Bedford House, Hanley, Staffordshire.
- 1848 †FORBES, JOHN GREGORY, 82, Oxford terrace, Hyde park. C. 1868-9. *Trans.* 3.
- 1852 †FORSTER, JOHN COOPER, *Vice-President*, Surgeon to Guy's Hospital; Examiner in Surgery at the University of London; 29, Upper Grosvenor street. C. 1868-9. S. 1873-5. V.P. 1877. *Pro.* 1.
- 1877 *FORTESCUE, GEORGE, M.B., late Surgeon to the Sydney Infirmary; Sydney, New South Wales.

Elected

- 1865 FOSTER, BALTHAZAR WALTER, M.D., Professor of Medicine at the Queen's College, Birmingham, and Physician to the Birmingham General Hospital; 16, Temple row, Birmingham.
- 1859 FOX, EDWARD LONG, M.D., Consulting Physician to the Bristol Royal Infirmary, and Lecturer on Medicine at the Bristol School of Medicine; Church House, Clifton, Gloucestershire.
- 1877 FOX, TILBURY, M.D., Physician to the Skin Department of University College Hospital; 14, Harley street.
- 1858 FOX, WILSON, M.D., F.R.S., Physician-Extraordinary to H.M. the Queen; Physician in Ordinary to H.R.H. the Duke of Edinburgh; Holme Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; Examiner in Medicine at the University of London; 67, Grosvenor street. C. 1875-6. *Trans.* 3.
- 1871 FRANK, PHILIP, M.D., Cannes, France.
- 1843 FRASER, PATRICK, M.D. C. 1866.
- 1868 FREEMAN, WILLIAM HENRY, 21, St. George's square, South Belgravia.
- 1836 †FRENCH, JOHN GEORGE, 10, Cunningham place, Maida hill. C. 1852-3.
- 1876 FURNER, WILLOUGHBY, 111, King's road, Brighton.
- 1864 *GAIRDNER, WILLIAM TENNANT, M.D., Physician in Ordinary to H.M. the Queen in Scotland; Professor of the Practice of Medicine in the University of Glasgow; Physician to the Glasgow Royal Infirmary; 225, St. Vincent street, Glasgow.
- 1874 GALABIN, ALFRED LEWIS, M.A., M.D., Assistant Obstetric Physician to, and Lecturer on Midwifery and the Diseases of Women at, Guy's Hospital; Assistant-Physician to the Hospital for Sick Children; 14, St. Thomas's street, Southwark. *Trans.* 2.
- 1865 GANT, FREDERICK JAMES, Surgeon to the Royal Free Hospital, 16, Connaught square, Hyde park. *Trans.* 2.

Elected

- 1867 GARLAND, EDWARD CHARLES, L.R.C.P. Edin., Yeovil, Somerset.
- 1867 GARLIKE, THOMAS W., Highfield, 126, Tulse hill, Brixton.
- 1854 GARROD, ALFRED BARING, M.D., F.R.S., Consulting Physician to King's College Hospital; 10, Harley street, Cavendish square. C. 1867. *Trans.* 8.
- 1851 †GASKOIN, GEORGE, Surgeon to the British Hospital for Diseases of the Skin; 7, Westbourne park. C. 1875-6. *Trans.* 1.
- 1819 GAULTER, HENRY.
- 1848 GAY, JOHN, Senior Surgeon to the Great Northern Hospital, and Consulting Surgeon to the Asylum for Idiots; 10, Finsbury place south. C. 1874-5.
- 1866 GEE, SAMUEL JONES, M.D., Assistant-Physician to St. Bartholomew's Hospital; Physician to the Hospital for Sick Children; 54, Harley street, Cavendish square. *Trans.* 1.
- 1821 *GEORGE, RICHARD FRANCIS, 1, South Parade, Weston-super-Mare.
- 1877 GODLEE, RICKMAN JOHN, Assistant-Surgeon to Charing-Cross Hospital; Assistant-Surgeon to University College Hospital; and Demonstrator of Anatomy at University College; 22, Henrietta street, Cavendish square.
- 1870 GODSON, CLEMENT, M.D., Assistant-Physician-Accoucheur to St. Bartholomew's Hospital; Physician to the Samaritan Free Hospital; 8, Upper Brook street, Grosvenor square.
- 1867 GOODEVE, EDWARD, M.B., Hon. Physician to H.M. the Queen; late Surgeon-Major, H.M.'s Bengal Army; Drimagh, Stoke Bishop, near Bristol.
- 1851 GOODFELLOW, STEPHEN JENNINGS, M.D., Consulting Physician to the Middlesex Hospital; Swinnerton Lodge, near Dartmouth, Devon. C. 1864-5. *Trans.* 2.
- 1877 GOULD, ALFRED PEARCE, M.S., Lecturer on Anatomy at the Westminster Hospital; 93, Gower street, Bedford square.
- 1873 GOWERS, WILLIAM RICHARD, M.D., Assistant Professor of Clinical Medicine at University College, and Assistant-Physician to University College Hospital; 50, Queen Anne street. *Trans.* 4.

Elected

- 1851 GOWLLAND, PETER YEAMES, Surgeon to St. Mark's Hospital; Surgeon-Major Hon. Artillery Company; 34, Finsbury Square.
- 1846 GREAM, GEORGE THOMPSON, M.D., Physician-Accoucheur to H.R.H. the Princess of Wales; Heathfield, Ringwood, Hants. C. 1863.
- 1868 GREEN, T. HENRY, M.D., Physician to, and Lecturer on Pathology at, Charing Cross Hospital; Assistant-Physician to the Hospital for Consumption, Brompton; 74, Wimpole street, Cavendish square.
- 1875 GREENE, WILLIAM T., M.A., M.D., 218, Old Kent road.
- 1875 GREENFIELD, W. S., M.D., Assistant-Physician to, and Lecturer on Morbid Anatomy at, St. Thomas's Hospital; 93, Wimpole street, Cavendish square.
- 1843 †GREENHALGH, ROBERT, M.D., Consulting Physician to the Samaritan Free Hospital for Women and Children, and to the City of London Lying-in Hospital; 72, Grosvenor street. C. 1871-2. *Trans.* 1.
- 1860 GREENHOW, EDWARD HEADLAM, M.D., F.R.S., Physician to the Middlesex Hospital; and Consulting Physician to the Western General Dispensary; 14A, Manchester square. C. 1876-7. *Trans.* 3.
- 1868 GRIGG, WILLIAM CHAPMAN, M.D., Assistant Obstetric Physician to the Westminster Hospital; Physician to the In-Patients, Queen Charlotte's Lying-in-Hospital; Assistant-Physician to the Victoria Hospital for Children; 6, Curzon street, Mayfair.
- 1852 GROVE, JOHN, Spring Grove, Hampton, Middlesex.
- 1860 GUENEAU DE MUSSY, HENRI, M.D.; 15, Rue du Cirque, Paris.
- 1849 GULL, SIR WILLIAM WITHEY, Bart., M.D., D.C.L., F.R.S., Physician-Extraordinary to the Queen; Member of the Senate of the University of London; Consulting Physician to Guy's Hospital; 74, Brook street, Grosvenor square. C. 1864. V.P. 1874. *Trans.* 4.

Elected

- 1837 GULLY, JAMES MANBY, M.D.
- 1854 HABERSHON, SAMUEL OSBORNE, M.D., Physician to Guy's Hospital; 70, Brook street, Grosvenor square. S. 1867. C. 1869-70. *Trans.* 3.
- 1849 HAILEY, HAMMETT, F.L.S., Tickford Lodge, Newport Pagnell, Bucks.
- 1870 HAMILTON, ROBERT, Surgeon to the South Hospital, Liverpool; 1 Prince's road, Liverpool.
- 1838 †HANCOCK, HENRY, Consulting Surgeon to the Charing Cross Hospital, and to the Royal Westminster Ophthalmic Hospital; Standen House, Chute, Wilts. C. 1851. V.P. 1869.
- 1874 HARDIE, GORDON KENMURE, M.D., Deputy Inspector General of Hospitals; 13, Sussex place, Onslow gardens, and Duff House, Banff, N.B.
- 1836 HARDING, JOHN FOSSE, Ulverstone House, Uckfield, Sussex. C. 1858-9.
- 1856 HARE, CHARLES JOHN, M.D., late Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 57, Brook street, Grosvenor square. C. 1873-4.
- 1857 HARLEY, GEORGE, M.D., F.R.S. [25, Harley street, Cavendish square]. C. 1871-2. *Trans.* 1. *Sci. Com.* 2.
- 1864 HARLEY, JOHN, M.D., F.L.S., *Secretary*, Assistant-Physician to, and Lecturer on Physiology at, St. Thomas's Hospital; 39, Brook street, Grosvenor square. S. 1875-7. *Trans.* 7.
- 1866 HARPER, PHILIP H., 30, Cambridge street, Hyde park.
- 1859 HARRIS, FRANCIS, M.D., F.L.S., 24, Cavendish square.
- 1872 HARRIS, WILLIAM H., M.D., Professor of Midwifery and Diseases of Women and Children, Madras Medical College, Madras.
- 1870 HARRISON, REGINALD, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Surgery at the School of Medicine; 38, Rodney street, Liverpool.

Elected

- 1854 HAVILAND, ALFRED, Medical Officer of Health for the combined Districts of Northamptonshire; Northampton.
- 1870 HAWARD, J. WARRINGTON, Assistant-Surgeon to St. George's Hospital; Surgeon to the Hospital for Sick Children; 5, Montagu street, Portman square. *Trans.* 1.
- 1828 †HAWKINS, CÆSAR HENRY, F.R.S., Sergeant-Surgeon to H.M. the Queen, and Consulting Surgeon to St. George's Hospital; 26, Grosvenor street. C. 1830-1, 1860. V.P. 1838-9. T. 1841-4. P. 1855-6. *Trans.* 12.
- 1838 †HAWKINS, CHARLES, Inspector of Anatomical Schools in London; 27, Savile row, Burlington gardens. C. 1846-7. S. 1850. V.P. 1858. T. 1861-2. *Trans.* 2.
- 1848 HAWKSLEY, THOMAS, M.D., Consulting Physician to the Margaret street Dispensary for Consumption and Diseases of the Chest; 17, Cheyne walk, Chelsea.
- 1875 HAYES, THOMAS CRAWFORD, M.D., Assistant-Physician-Accoucheur and Assistant-Physician for Diseases of Women and Children to King's College Hospital; 17, Clarges street, Piccadilly.
- 1860 HAYWARD, HENRY HOWARD, Surgeon Dentist to, and Lecturer on Dental Surgery at, St. Mary's Hospital; 38, Harley street, Cavendish square.
- 1861 HAYWARD, WILLIAM HENRY, Church House, Oldbury, Worcestershire.
- 1848 *HEALE, JAMES NEWTON, M.D., Medecroft, Winchester, Hants.
- 1865 HEATH, CHRISTOPHER, Holme Professor of Clinical Surgery in University College, London; and Surgeon to University College Hospital; 36, Cavendish square.
- 1850 HEATON, GEORGE, M.D., Boston, U.S.
- 1874 *HEATON, JOHN DEAKIN, M.D., Senior Physician to the Leeds General Infirmary, and Lecturer on Medicine at the Leeds School of Medicine; Claremont, Leeds.
- 1821 HERBERSKI, VINCENT, M.D., Professor of Medicine in the University of Wilna.

Elected

- 1843 HEWETT, PRESCOTT GARDNER, F.R.S., Serjeant-Surgeon-Extraordinary to H.M. the Queen ; Surgeon in Ordinary to H.R.H. the Prince of Wales ; Consulting Surgeon to St. George's Hospital ; Corresponding Member of the Academy of Medicine, and of the "Société de Chirurgie," Paris ; 1, Chesterfield street, Mayfair. C. 1859. V.P. 1866-7. *Trans.* 7. *Sci. Com.*
- 1855 HEWITT, GRAILY, M.D., Professor of Midwifery in University College, London, and Obstetric Physician to University College Hospital ; 36, Berkeley square. C. 1876.
- 1872 HEYN, JULIUS CHARLES WILLIAM, M.D., 42, Westbourne terrace, Hyde park.
- 1873 HIGGENS, CHARLES, Assistant Ophthalmic Surgeon to Guy's Hospital ; 38, Brook street, Grosvenor square.
- 1862 HILL, M. BERKELEY, M.B. Lond., Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital ; Surgeon to the Lock Hospital ; 55, Wimpole street, Cavendish square.
- 1867 HILL, SAMUEL, M.D., 22, Mecklenburgh square.
- 1841 †HILTON, JOHN, F.R.S., Surgeon-Extraordinary to H.M. the Queen ; Consulting Surgeon to Guy's Hospital ; Consulting Surgeon to the Royal General Dispensary, St. Pancras ; 10, New Broad street, City. C. 1851. V.P. 1863-4. *Trans.* 4.
- 1859 HIRD, FRANCIS, Surgeon to the Charing Cross Hospital ; 13, Old Burlington street.
- 1861 *HOFFMEISTER, WILLIAM CARTER, M.D., Surgeon to H.M. the Queen in the Isle of Wight ; Clifton House, Cowes, Isle of Wight.
- 1872 HOGG, FRANCIS ROBERTS, M.D., India.
- 1843 †HOLDEN, LUTHER, Surgeon to St. Bartholomew's Hospital ; Consulting Surgeon to the Metropolitan Dispensary ; Surgeon to the Foundling Hospital ; 65, Gower street, Bedford square. C. 1859. L. 1865. V.P. 1874.

Elected

- 1868 HOLLIS, WILLIAM AINSLIE, M.A., M.B., Camb., Assistant-Physician to the Sussex County Hospital; 10, Old Steyne, Brighton.
- 1861 HOLMAN, WILLIAM HENRY, M.B. Lond., 68, Adelaide road, South Hampstead.
- 1856 HOLMES, TIMOTHY, M.A. Camb., *Librarian*, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; Surgeon in Chief to the Metropolitan Police Force; 18, Great Cumberland place, Hyde park. C. 1869-70. L. 1873-7. *Trans.* 6. *Sci. Com.*
- 1846 †HOLT, BARNARD WIGHT, Consulting Surgeon to, and Lecturer on Clinical Surgery at, the Westminster Hospital; Medical Officer of Health for Westminster; 14, Savile row, Burlington gardens. C. 1862-3.
- 1846 HOLTHOUSE, CARSTEN, 15, George street, Hanover square, and Balham hill house. C. 1863.
- 1865 HOWARD, BENJAMIN, M.D., late Lecturer on Operative Surgery, and Surgeon to the Long Island College Hospital, New York.
- 1865 HOWARD, EDWARD, M.D.
- 1874 HOWSE, HENRY GREENWAY, M.S. Lond., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children; 10, St. Thomas's street, Southwark. *Trans.* 2.
- 1877 *HUDSON, ROBERT SAMUEL, M.D., 58, West-end, Redruth, Cornwall.
- 1857 HULKE, JOHN WHITAKER, F.R.S., *Secretary*, Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 10, Old Burlington street. C. 1871-2. S. 1876-7. *Trans.* 7. *Sci. Com.*
- 1857 HULME, EDWARD CHARLES, Woodbridge road, Guildford. *Trans.* 1.
- 1844 †HUMBY, EDWIN, M.D., 83, Hamilton terrace, St. John's wood. C. 1866-7.
- 1855 HUMPHRY, GEORGE MURRAY, M.D., F.R.S., Surgeon to Addenbrooke's Hospital; Professor of Human Anatomy and Physiology in the Cambridge University Medical School; Cambridge. *Trans.* 5.

Elected

- 1866 HUNTER, CHARLES, Ben Rhydding, by Leeds.
- 1873 HUNTER, WILLIAM GUYER, M.D., Principal of, and Professor of Medicine in, Grant Medical College, Bombay ; Surgeon-Major, Bombay Army, Bombay.
- 1849 HUSSEY, EDWARD LAW, Senior Surgeon to the Radcliffe Infirmary, and Consulting Surgeon to the County Lunatic Asylum and the Warneford Asylum ; 8, St. Aldate's, Oxford. *Trans.* 1.
- 1856 HUTCHINSON, JONATHAN, Surgeon to the London Hospital ; Surgeon to the Royal London Ophthalmic Hospital, Moorfields, and to the Hospital for Diseases of the Skin ; 15, Cavendish square. C. 1870. *Trans.* 6. *Pro.* 2.
- 1820 HUTCHINSON, WILLIAM, M.D.
- 1840 †HUTTON, CHARLES, M.D., Consulting Physician to the General Lying-in Hospital ; 26, Lowndes street, Belgrave square. C. 1858-9.
- 1866 ILES, FRANCIS HENRY WILSON, M.D., Watford, Herts.
- 1847 IMAGE, WILLIAM EDMUND, Consulting Surgeon to the Suffolk General Hospital ; Bury St. Edmund's, Suffolk. *Trans.* 1.
- 1856 INGLIS, CORNELIUS, M.D., Athenæum Club, Pall Mall.
- 1876 IRVINE, JAMES PEARSON, M.D., Assistant Physician to, and Lecturer on Forensic Medicine at, the Charing Cross Hospital ; 3, Mansfield street, Cavendish square.
- 1871 JACKSON, J. HUGHLINGS, M.D., Physician to the London Hospital ; Physician to the National Hospital for the Paralysed and Epileptic ; 3, Manchester square.
- 1841 †JACKSON, PAUL, 24, Wimpole street, Cavendish square. C. 1862.
- 1868 JACKSON, THOMAS CARR, Surgeon to the Great Northern Hospital, and Surgeon to the National Orthopædic Hospital ; 91, Harley street, Cavendish square.
- 1863 JACKSON, THOMAS VINCENT, Surgeon to the South Staffordshire General Hospital ; Darlington st., Wolverhampton.

Elected

- 1841 JACOBOVICS, MAXIMILIAN MORITZ, M.D., Vienna.
- 1825 JAMES, JOHN B., M.D.
- 1840 *JENKS, GEORGE SAMUEL, M.D., 18, Circus, Bath.
- 1851 JENNER, SIR WILLIAM, Bart., M.D., K.C.B., D.C.L., F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; Member of the Senate of the University of London; 63, Brook street, Grosvenor square. C. 1864. V.P. 1875. *Trans.* 3.
- 1851 JOHNSON, EDMUND CHARLES, Corresponding Member of the Medical and Philosophical Society of Florence, and of "l'Institut Genevois."
- 1847 JOHNSON, GEORGE, M.D., F.R.S., Physician to King's College Hospital; Member of the Senate of the University of London; 11, Savile row, Burlington gardens. C. 1862-3. V.P. 1870. *Trans.* 10.
- 1868 JOHNSTON, WILLIAM, M.D., 44, Princes square, Hyde park.
- 1848 JOHNSTONE, ATHOL ARCHIBALD WOOD, Consulting Surgeon to the Brighton Hospital for Sick Children, St. Moritz House, 61, Dyke road, Brighton. *Trans.* 1.
- 1862 JONES, CHARLES HANDFIELD, M.B., F.R.S., Physician to, and Lecturer on Clinical Medicine at, St. Mary's Hospital; 49, Green street, Grosvenor square.
- 1876 JONES, LESLIE, M.D., 3, Brighton Parade, Blackpool, Lancashire.
- 1875 *JONES, PHILIP SYDNEY, M.D., Consulting Surgeon to the Sydney Infirmary; Examiner in Medicine, Sydney University. [Agents: Messrs. D. Jones & Co., 1, Gresham buildings, Basinghall street.]
- 1837 †JONES, THOMAS WILLIAM, M.D., Bylocks, Enfield Highway. C. 1858.
- 1859 JONES, WILLIAM PRICE, M.D., Claremont road, Surbiton, Kingston.

Elected

- 1865 JORDAN, FURNEAUX, Surgeon to the Queen's Hospital, and Professor of Surgery at the Queen's College, Birmingham; 22, Colmore row, Birmingham.
- 1816 *KAUFFMANN, GEORGE HERMANN, M.D., Hanover.
- 1872 KELLY, CHARLES, M.D., Medical Officer of Health for the West Sussex Combined Sanitary District, Worthing, Sussex.
- 1848 *KENDELL, DANIEL BURTON, M.D., Heath House, Wakefield, Yorkshire.
- 1847 KEYSER, ALFRED, King's Hill, Berkhamstead.
- 1857 KIALLMARK, HENRY WALTER, 66, Princes square, Bayswater.
- 1851 KINGDON, JOHN ABERNETHY, Surgeon to the City of London Truss Society, and to the City Dispensary; 2, New Bank buildings, Lothbury. C. 1866-7. V.P. 1872-3. *Trans.* 1. *Sci. Com.*
- 1876 *KOCH, EDWIN LAWSON, M.D., Principal, Medical School of Ceylon; Colombo, Ceylon. [Agents: Messrs. Henry S. King & Co., 65, Cornhill.]
- 1855 LANE, JAMES ROBERT, Surgeon to, and Lecturer on Surgery at, St. Mary's Hospital; Surgeon to the Lock Hospital; 49, Norfolk square, Hyde park. C. 1870. *Trans.* 1.
- 1840 †LANE, SAMUEL ARMSTRONG, Consulting Surgeon to St. Mary's Hospital and to the Lock Hospital; 49, Norfolk square, Hyde park. C. 1849-50. V.P. 1865.
- 1865 LANGTON, JOHN, Assistant-Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the City of London Truss Society; 2, Harley street, Cavendish square.
- 1873 *LARCHER, O., M.D., Laureate of the Institute of France, of the Medical Faculty, and Academy of Paris, &c.; 97, Rue de Passy, Passy, Paris.
- 1841 *LASHMAR, CHARLES, M.D., 83, North End, Croydon, Surrey.

Elected

- 1862 LATHAM, PETER WALLWORK, M.A., M.D., Downing Professor of Medicine, Cambridge University; Physician to Addenbrooke's Hospital, Cambridge; 17, Trumpington street, Cambridge.
- 1816 LAWRENCE, G. E.
- 1843 *LEACH, JESSE, Moss Hall, Heywood, Lancashire.
- 1868 LEARED, ARTHUR, M.D., Senior Physician to the Great Northern Hospital; 12, Old Burlington street.
- 1843 †LEE, HENRY, Surgeon to, and Lecturer on Clinical Surgery at, St. George's Hospital; 9, Savile row, Burlington gardens. C. 1856-7. L. 1863-4. V. P. 1868-9. *Trans.* 13. *Pro.* 2. *Sci. Com.*
- 1869 LEGG, JOHN WICKHAM, M.D., Physician to Casualty Department and Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital; 47, Green street, Park lane. *Trans.* 2.
- 1836 LEIGHTON, FREDERICK, M.D.
- 1872 LIEBBEICH, RICHARD, Ophthalmic Surgeon and Lecturer on Ophthalmic Surgery at St. Thomas's Hospital; 16, Albemarle street, Piccadilly.
- 1806 LIND, JOHN, M.D.
- 1872 *LITTLE, DAVID, M.D., Surgeon to the Royal Eye Hospital, Manchester; 21, St. John's street, Manchester.
- 1871 LITTLE, LOUIS STROMEYER, Shanghai, China.
- 1870 LIVINGSTON, JOHN, M.D., New Barnet, Hertfordshire.
- 1819 LLOYD, ROBERT, M.D.
- 1820 LOCHER, J. G., M.C.D., Town Physician of Zurich. *Trans.* 2.
- 1860 LONGMORE, THOMAS, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-General, Army Medical Staff, and Professor of Military Surgery, Army Medical School, Netley, Southampton; Woolston Lawn, Woolston, Hants. *Trans.* 2.

Elected

- 1836 LÖWENFELD, JOSEPH S., M.D., Berbice.
- 1871 LOWNDS, THOMAS MACKFORD, M.D., late Professor of Anatomy and Physiology at Grant Medical College, Bombay; Egham Hill, Surrey.
- 1852 LUKE, JAMES, F.R.S., Consulting Surgeon to the London Hospital; Woolley Lodge, Maidenhead Thicket, Berks. C. 1858. *Trans.* 4.
- 1857 LYON, FELIX WILLIAM, M.D., 4, Graham street, Edinburgh.
- 1867 MABERLY, GEORGE FREDERICK, Leamington, Warwickshire.
- 1873 MACCARTHY, JEREMIAH, M.A., Surgeon to, and Lecturer on Physiology at, the London Hospital; 26, Finsbury square.
- 1867 MAC CORMAC, WILLIAM, M.A., Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; 13, Harley street. *Trans.* 1.
- 1862 *M'DONNELL, ROBERT, M.D., F.R.S., Surgeon to Steevens' Hospital; 14, Lower Pembroke street, Dublin. *Trans.* 2.
- 1846 M'EWEN, WILLIAM, M.D., Surgeon to Chester Castle; 27, Nicholas street, Chester.
- 1866 MACGOWAN, ALEXANDER THORBURN, Kingswood park, near Bristol.
- 1823 †MACILWAIN, GEORGE, Consulting Surgeon to the Finsbury Dispensary, Matching, Harlow, Essex. C. 1829-30. V.P. 1848. *Trans.* 1.
- 1822 MACINTOSH, RICHARD, M.D.
- 1859 *M'INTYRE, JOHN, M.D., Odiham, Hants.
- 1873 MACKELLAR, ALEXANDER OBERLIN, M.S.I., Assistant Surgeon, St. Thomas's Hospital; Albert Embankment, Westminster Bridge.
- 1876 MACKEY, EDWARD, M.D., 54, Inverness terrace, Bayswater.
- 1854 *MACKINDER, DRAPER, M.D., Consulting Surgeon to the Dispensary, Gainsborough, Lincolnshire.

Elected

- 1860 MACLEAN, JOHN, M.D., 24, Portman street, Portman square.
- 1849 MACLURE, DUNCAN MACLACHLAN, M.B., Lecturer on Physiology at the Westminster Hospital; Assistant-Physician to the National Hospital for the Paralysed and Epileptic; 34, Harley Street, Cavendish square.
- 1876 MACNAMARA, CHARLES, Surgeon to the Westminster Hospital; Surgeon Major Bengal Medical Service; late Examiner in Surgery at the Calcutta University; 13, Grosvenor street.
- 1842 MACNAUGHT, JOHN, M.D., 74, Huskisson street, Liverpool.
- 1876 MALLAM, BENJAMIN, Percy Villa, 316, Camden road.
- 1855 MARCET, WILLIAM, M.D., F.R.S.; Villa Bianca, Cannes. C. 1871. *Trans.* 3. *Sci. Com.*
- 1867 MARSH, F. HOWARD, Assistant-Surgeon to St. Bartholomew's Hospital; 36, Bruton street, Berkeley square. *Trans.* 2.
- 1838 MARSH, THOMAS PARR, M.D.
- 1851 MARSHALL, JOHN, F.R.S., Professor of Anatomy to the Royal Academy of Arts; Professor of Surgery in University College, London, and Surgeon to University College Hospital; 10, Savile row, Burlington gardens. C. 1866. V.P. 1875-76. *Trans.* 2.
- 1864 MASON, FRANCIS, Surgeon to, and Lecturer on Anatomy at, St. Thomas's Hospital; 5, Brook street, Grosvenor square. *Trans.* 1.
- 1839 MEADE, RICHARD HENRY, Consulting Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans.* 1.
- 1870 MEADOWS, ALFRED, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Mary's Hospital; 27, George street, Hanover square.
- 1865 MEDWIN, AARON GEORGE, M.D., Dental Surgeon to the Royal Kent Dispensary, 11, Montpellier row, Blackheath, Kent.
- 1867 MEREDYTH, COLOMIATI, M.D., 10, George street, Hanover square.

Elected

- 1874 MERRIMAN, JOHN J., 45, Kensington square.
- 1852 MERRYWEATHER, JAMES, Consulting Surgeon to the National Dental Hospital; 25, Brook street, Grosvenor square.
- 1847 MERYON, EDWARD, M.D., F.G.S., 14, Clarges street, Piccadilly. L. 1859-60. C. 1864-5. V.P. 1868-9. *Trans.* 2.
- 1815 MEYER, AUGUSTUS, M.D., St. Petersburg.
- 1840 MIDDLEMORE, RICHARD, Consulting Surgeon to the Birmingham Eye Hospital; 19, Temple row, Birmingham.
- 1854 MIDDLESHP, EDWARD ARCHIBALD.
- 1873 MILNER, EDWARD, Surgeon to the Lock Hospital; 32, New Cavendish street, Portland place.
- 1863 MONRO, HENRY, M.D., Physician to St. Luke's Hospital; 13, Cavendish square. C. 1868.
- 1844 †MONTEFIORE, NATHANIEL, 36, Hyde park gardens.
- 1836 MOORE, GEORGE, M.D., Hastings.
- 1873 MOORE, NORMAN, M.D., Warden of the College and Lecturer on Comparative Anatomy, St. Bartholomew's Hospital; the College, St. Bartholomew's Hospital.
- 1861 MOREHEAD, CHARLES, M.D., Hon. Surgeon to H.M. the Queen; Deputy-Inspector General of Hospitals; 11, North manor place, Edinburgh.
- 1857 MORGAN, JOHN, 3, Sussex place, Hyde park gardens. *Trans.* 1.
- 1861 MORGAN, JOHN EDWARD, M.D., Physician to the Manchester Royal Infirmary, and Professor of Medicine in the Owens College, Manchester; 1, St. Peter's square, Manchester.
- 1874 MORRIS, HENRY, M.A. Lond., Senior Assistant-Surgeon to, and Lecturer on Anatomy at, the Middlesex Hospital; 2, Mansfield street, Portland place. *Trans.* 2.
- 1851 MOUAT, FREDERIC JOHN, M.D., Deputy Inspector-General of Hospitals; Medical Inspector to the Local Government Board; and Member of the Senate of the University of Calcutta; 12, Durham villas, Kensington.

Elected

- 1868 MOXON, WALTER, M.D., F.L.S., Physician to, and Lecturer on Clinical Medicine at, Guy's Hospital; 6, Finsbury Circus. *Trans.* 1.
- 1856 MURCHISON, CHARLES, M.D., LL.D. Edinb., F.R.S., *Librarian*, Physician to, and Lecturer on Clinical Medicine at, St. Thomas's Hospital, Consulting Physician to the London Fever Hospital; Examiner in Medicine at the University of London; 79, Wimpole street, Cavendish square. C. 1870-71. L. 1877. *Trans.* 3.
- 1875 MURPHY, WILLIAM KIRKPATRICK, M.A., M.D., 29, Queen Anne street, Cavendish square.
- 1873 MURRAY, IVOR, M.D., F.R.S. Ed., 8, Huntress Row, Scarborough.
- 1863 MYERS, ARTHUR B. R., Surgeon to the 1st Battalion Coldstream Guards; Hospital, Vincent square, Westminster.
- 1876 NAPIER, WILLIAM DONALD, 22, George street, Hanover square, W.
- 1870 NEILD, JAMES EDWARD, M.D., Lecturer on Forensic Medicine in the University of Melbourne; 166, Collins street east, Melbourne, Victoria.
- 1835 †NELSON, THOMAS ANDREW, M.D., 10, Nottingham terrace, York gate, Regent's park.
- 1843 †NEWTON, EDWARD [4, Upper Wimpole street]. C. 1863-4.
- 1868 NICHOLLS, JAMES, M.D., Duke street, Chelmsford, Essex.
- 1849 NORMAN, HENRY BURFORD, Portland Lodge, Southsea, Hants.
- 1847 *NOURSE, WILLIAM EDWARD CHARLES, late Surgeon to the Brighton Children's Hospital; 11, Marlborough place, Brighton.
- 1849 NOVERRE, ARTHUR, 16, Park street, Grosvenor square. C. 1870-71.
- 1864 NUNN, THOMAS WILLIAM, Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.
- 1870 NUNNELEY, FREDERICK BARHAM, M.D. *Trans.* 2.

Elected

- 1847 O'CONNOR, THOMAS, March, Cambridgeshire.
- 1843 †O'CONNOR, WILLIAM, M.D., Senior Physician to the Royal Free Hospital; 30, Upper Montagu street, Montagu square.
- 1858 OGLE, JOHN WILLIAM, M.D., Consulting-Physician to St. George's Hospital; 30, Cavendish square. C. 1873. *Trans.* 4.
- 1855 *OGLE, WILLIAM, M.A., M.D., Physician to the Derby Infirmary; 98, Friar Gate, and The Elms, Derby.
- 1860 OGLE, WILLIAM, M.D., Medical Officer of Health for East Hertfordshire; 10, Gordon street, Gordon square. S. 1868-70. C. 1876-7. *Trans.* 4.
- 1870 OLDHAM, CHARLES FREDERIC, India [Agents: Messrs. Grindlay and Co., 55, Parliament Street].
- 1871 *O'NEILL, WILLIAM, M.D., Physician to the Lincoln Lunatic Hospital, Lincoln.
- 1873 ORD, WILLIAM MILLER, M.B., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 7, Brook street, Hanover square. *Trans.* 3.
- 1875 OSBORN, SAMUEL,; 17, Gresham park, Brixton.
- 1874 PAGE, HERBERT WILLIAM, M.B., M.C., Assistant Surgeon and Lecturer on the Practice of Surgery at St. Mary's Hospital; 28, New Cavendish street.
- 1847 *PAGE, WILLIAM BOUSFIELD, Surgeon to the Cumberland Infirmary, Carlisle. *Trans.* 2.
- 1840 †PAGET, SIR JAMES, Bart., D.C.L., LL.D., F.R.S., Sergeant-Surgeon to H.M. the Queen; Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital; Member of the Senate of the University of London; 1, Harewood place, Hanover square. C. 1848-49. V.P. 1861. T. 1867. P. 1875-76. *Trans.* 10. *Sci. Com.*
- 1858 *PALEY, WILLIAM, M.D., Physician to the Ripon Dispensary; Ripon, Yorkshire.
- 1847 PARKER, NICHOLAS, M.D., Paris.

Elected

- 1873 PARKER, ROBERT WILLIAM, Assistant-Surgeon East London Children's Hospital ; 8, Old Cavendish-street. *Trans.* 1.
- 1841 PARKIN, JOHN, M.D. Rome ; 10, Margaret street, Cavendish square.
- 1865 PAVY, FREDERICK WILLIAM, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital ; 35, Grosvenor street.
- 1869 PAYNE, JOSEPH FRANK, M.B., Assistant-Physician to, and Lecturer on Forensic Medicine at, St. Thomas's Hospital ; 78, Wimpole street.
- 1845 †PEACOCK, THOMAS BEVILL, M.D., Consulting Physician to St. Thomas's Hospital ; Consulting Physician to the City of London Hospital for Diseases of the Chest, Victoria Park ; 20, Finsbury circus. S. 1855-6. V.P. 1867. C. 1869. *Trans.* 2.
- 1856 PEIRCE, RICHARD KING, 94, Addison road, Kensington.
- 1830 PELECHIN, CHARLES P., M.D., St. Petersburg.
- 1855 *PEMBERTON, OLIVER, Surgeon to the Birmingham General Hospital, and Professor of Surgery at the Queen's College, Birmingham ; 18, Temple row, Birmingham. *Trans.* 1.
- 1874 PENHALL, JOHN THOMAS, 5, Eversfield place, St. Leonard's, Sussex.
- 1870 PERRIN, JOHN BESWICK, Medical Tutor and Demonstrator of Practical and Surgical Anatomy, Owen's College ; 51, Nelson street, Manchester.
- 1852 PHILLIPS, RICHARD, 27, Leinster square, Bayswater. C. 1877.
- 1846 PHILP, FRANCIS RICHARD, M.D. [Colby House, Kensington.]
- 1867 PICK, THOMAS PICKERING, Assistant-Surgeon to, and Lecturer on Anatomy at, St. George's Hospital ; 13, South Eaton place, Eaton square. *Sci. Com.*
- 1841 †PITMAN, HENRY ALFRED, M.D., Consulting Physician to St. George's Hospital, and to the Royal General Dispensary, St. Pancras ; 28, Gordon square. L. 1851-3. C. 1861-2. T. 1863-8. V.P. 1870-1.

Elected

- 1871 POLLOCK, ARTHUR JULIUS, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, Charing Cross Hospital; Physician to the Foundling Hospital; 85, Harley street, Cavendish square.
- 1845 †POLLOCK, GEORGE DAVID, Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Surgeon to St. George's Hospital; 36, Grosvenor street. C. 1856-7. L. 1859-62. V.P. 1870-1. *Trans.* 4.
- 1865 POLLOCK, JAMES EDWARD, M.D., Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square.
- 1871 POORE, GEORGE VIVIAN, M.D., Assistant-Physician to University College Hospital; Physician to the Royal Infirmary for Children and Women, Waterloo road; 30, Wimpole street.
- 1843 POPE, CHARLES, M.D., The Rectory, East Harptree, Bristol.
- 1846 POTTER, JEPHSON, M.D., F.L.S.
- 1842 POWELL, JAMES, M.D.
- 1867 POWELL, RICHARD DOUGLAS, M.D., Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 15, Henrietta street, Cavendish square. *Trans.* 1.
- 1867 POWER, HENRY, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. Bartholomew's Hospital; 37A, Gt. Cumberland place, Hyde park. *Sci. Com.*
- 1857 PRIESTLEY, WILLIAM OVEREND, M.D., Physician-Accoucheur to H.R.H. the Princess Louis of Hesse; Consulting Physician-Accoucheur to King's College Hospital, and to the St. Marylebone Infirmary; 17, Hertford street, Mayfair. C. 1874-5. *Sci. Com.*
- 1869 PULLAR, ALFRED, M.D., Surgeon to the Kensington Dispensary; 1, Pembridge place, Bayswater.
- 1874 PURVES, WILLIAM LAIDLAW, M.D., Aural Surgeon to Guy's Hospital; 9, Upper Wimpole street, Cavendish square. *Trans.* 1.
- 1850 QUAIN, RICHARD, M.D., F.R.S., Consulting Physician to the Hospital for Consumption, Brompton; Member of the Senate of the University of London; 67, Harley street, Cavendish square. C. 1866-7. *Trans.* 1. *Sci. Com.*

Elected

- 1835 †QUAIN, RICHARD, F.R.S., Surgeon-Extraordinary to H.M. the Queen; Emeritus Professor of Clinical Surgery, University College, London, and Consulting Surgeon to the Eye Infirmary attached to the Hospital; 32, Cavendish square. C. 1838-9. L. 1846-8. T. 1851-3. V.P. 1856-7. *Trans.* 1. *Pro.* 2.
- 1852 RADCLIFFE, CHARLES BLAND, M.D., Consulting Physician to the Westminster Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 25, Cavendish square. C. 1867-8.
- 1871 RALFE, CHARLES HENRY, M.D., M.A., Physician to the Seamen's Hospital, Greenwich; 26, Queen Anne street, Cavendish square.
- 1857 RANKE, HENRY, M.D., 3, Sophienstrasse, Munich.
- 1854 RANSOM, WILLIAM HENRY, M.D., F.R.S., Physician to the Nottingham General Hospital; the Pavement, Nottingham.
- 1869 READ, THOMAS LAURENCE, 57, Gloucester road [11, Peter-sham terrace], Queen's gate, South Kensington.
- 1858 REED, FREDERICK GEORGE, M.D., 46, Hertford street, Mayfair. *Trans.* 1.
- 1821 REEDER, HENRY, M.D., Varick, Seneca County, New York, United States.
- 1857 REES, GEORGE OWEN, M.D., F.R.S., Consulting Physician to Guy's Hospital; 26, Albemarle street, Piccadilly. C. 1873. *Trans.* 1.
- 1869 REEVES, WILLIAM, 5, the Crescent, Carlisle.
- 1855 REYNOLDS, JOHN RUSSELL, M.D., F.R.S., Professor of the Principles and Practice of Medicine in University College, London, and Physician to University College Hospital; 38, Grosvenor street. C. 1870.
- 1865 RHODES, GEORGE WINTER, Surgeon to the Huddersfield Infirmary; Queen street south, Huddersfield.
- 1847 RICHARDS, SAMUEL, M.D., 36, Bedford square.

Elected

- 1852 RICHARDSON, CHRISTOPHER THOMAS, M.B.
- 1869 RICKARDS, WALTER, M.D., Physician to the Royal Free Hospital ; 8, Cavendish place, Cavendish square.
- 1845 †RIDGE, BENJAMIN, M.D., 8, Mount street, Grosvenor square.
- 1863 RINGER, SYDNEY, M.D., Professor of Materia Medica in University College, London, and Physician to University College Hospital ; Examiner in Materia Medica in the University of London ; 15, Cavendish place, Cavendish square. *Trans.* 4.
- 1871 RIVINGTON, WALTER, M.S., Surgeon to, and Lecturer on Anatomy at, the London Hospital ; 22, Finsbury square. *Trans.* 2.
- 1871 *ROBERTS, DAVID LLOYD, M.D., Physician to St. Mary's Hospital, Manchester ; 23, St. John's street, Deansgate, Manchester.
- 1852 ROBERTS, JOHN, M.R.C.P., the Park, Westow hill, Upper Norwood.
- 1857 ROBERTSON, JOHN CHARLES GEORGE, Medical Superintendent of the Cavan District Lunatic Asylum ; Monaghan, Ireland.
- 1873 ROBERTSON, WILLIAM H., M.D., Consulting Physician to the Devonshire Hospital and Buxton Bath Charity ; Buxton, Derbyshire.
- 1843 RODEN, WILLIAM M.D., Morningside, Kidderminster, Worcestershire.
- 1850 ROPER, GEORGE, M.D., Physician to the Royal Maternity Charity ; 6, West street, Finsbury circus.
- 1857 ROSE, HENRY COOPER, M.D., F.L.S., Surgeon to the Hampstead Dispensary ; Penrose House, Hampstead. *Trans.* 1.
- 1849 ROUTH, CHARLES HENRY FELIX, M.D., Physician to the Samaritan Free Hospital for Women and Children ; 52, Montagu square. *Trans.* 1.
- 1863 ROWE, THOMAS SMITH, M.D., Surgeon to the Royal Sea-Bathing Infirmary ; Cecil street, Margate, Kent.

Elected

- 1845 RUSSELL, JAMES, M.D., Physician to the Birmingham General-Hospital; 91, New Hall street, Birmingham.
- 1871 RUTHERFORD, WILLIAM, M.D., F.R.S., Professor of Physiology in the University of Edinburgh.
- 1856 SALTER, S. JAMES A., F.R.S., F.L.S., Dental Surgeon to, and Lecturer on Dental Surgery at, Guy's Hospital; 17, New Broad street, City. C. 1871. *Trans.* 2.
- 1849 †SANDERSON, HUGH JAMES, M.D., 26, Upper Berkeley street, Portman square. C. 1872-3.
- 1855 SANDERSON, JOHN BURDON, M.D., LL.D., F.R.S., Jodrell Professor of Human Physiology and Histology at University College, London; 49, Queen Anne street, Cavendish square. C. 1869-70. *Trans.* 2. *Sci. Com.* 2.
- 1867 SANDFORD, FOLLIOTT JAMES, M.D., Market Drayton, Shropshire.
- 1847 SANKEY, WILLIAM HENRY OCTAVIUS, M.D., Lecturer on Mental Diseases at University College, London; Sandywell park, Cheltenham.
- 1869 SANSOM, ARTHUR ERNEST, M.D., Assistant-Physician to the London Hospital; 30, Devonshire street, Portland place. *Trans.* 1.
- 1845 †SAUNDERS, EDWIN, Surgeon-Dentist to H.M. the Queen, and to H.R.H. the Prince of Wales; 13A, George street, Hanover square. C. 1872-3.
- 1834 SAUVAN, LUDWIG V., M.D., Warsaw.
- 1859 SAVORY, WILLIAM SCOVELL, F.R.S., Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Surgeon to Christ's Hospital; Examiner in Surgery at the University of London; 66, Brook street, Grosvenor square. C. 1871-2. *Trans.* 4. *Sci. Com.* 3.
- 1873 SCOTT, J. M. JOHNSTON, M.D., 14, College square, east, Belfast.
- 1861 *SCOTT, WILLIAM, M.D., Physician to the Huddersfield Infirmary; Waverley House, Huddersfield.

Elected

- 1863 SEDGWICK, WILLIAM, 12, Park place, Upper Baker street.
Trans. 2.
- 1877 SEMON, FELIX, M.D., 6, Chandos street, Cavendish square.
- 1875 SEMPLE, ROBERT HUNTER, M.D., Physician to the Bloomsbury Dispensary; 8, Torrington square.
- 1873 *SHAPTER, LEWIS, B.A., M.B., Physician to the Devon and Exeter Hospital; the Barnfield, Exeter.
- 1840 SHARP, WILLIAM, M.D., F.R.S., Horton House, Rugby.
Trans. 1.
- 1837 †SHARPEY, WILLIAM, M.D., F.R.S., LL.D., Member of the Senate of the University of London; 50, Torrington square. C. 1848-9. V.P. 1862.
- 1836 †SHAW, ALEXANDER, Consulting Surgeon to the Middlesex Hospital; 136, Abbey road, Kilburn. C. 1842. S. 1843-4. V.P. 1851-2. T. 1858-60. *Trans.* 4.
- 1848 *SHEARMAN, EDWARD JAMES, M.D., F.R.S. Edin., F.L.S., Consulting Physician to the Rotherham Hospital; Moorgate, Rotherham, Yorkshire.
- 1859 SIBLEY, SEPTIMUS WILLIAM, 4, Savile row. *Trans.* 4.
Sci. Com.
- 1848 SIEVEKING, EDWARD HENRY, M.D., Physician-Extraordinary to H.M. the Queen; Physician-in-Ordinary to H.R.H. the Prince of Wales; Physician to St. Mary's Hospital; 17, Manchester square. C. 1859-60. S. 1861-3. V.P. 1873-4. *Trans.* 2. *Sci. Com.*
- 1871 SILVER, ALEXANDER, M.D., Physician to, and Lecturer on Clinical Medicine at, Charing Cross Hospital; 2, Stafford street, Bond street.
- 1842 †SIMON, JOHN, C.B., D.C.L., F.R.S., Consulting Surgeon to St. Thomas's Hospital; 40, Kensington square. C. 1854-5. V.P. 1865. *Trans.* 1.
- 1865 SIMS, J. MARION, M.D., Surgeon to the New York State Women's Hospital; 267, Madison Avenue, New York.

Elected

- 1857 SIORDET, JAMES LEWIS, M.B., Villa Preti, Mentone, Nice.
- 1872 SMITH, GILBART, M.A., M.B., Physician to the Royal Hospital for Diseases of the Chest, City road; Visiting Physician to the Margaret Street Infirmary for Consumption; 68, Harley street, Cavendish square.
- 1866 SMITH, HEYWOOD, M.A. M.D. Oxon., Physician to the Hospital for Women; Physician to the British Lying-in Hospital; 2, Portugal street, Grosvenor square.
- 1835 SMITH, JOHN GREGORY, 23, Gloucester place, Greenwich.
- 1838 †SMITH, SPENCER, Surgeon to, and Lecturer on Clinical Surgery at, St. Mary's Hospital; 43, Oxford terrace, C. 1854. S. 1855-8. V.P. 1859-60. T. 1865.
- 1863 SMITH, THOMAS, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; Surgeon to the Hospital for Sick Children; 5, Stratford place, Oxford street. S. 1870-2. C. 1875-76. *Trans.* 3. *Sci. Com.*
- 1864 *SMITH, THOMAS HECKSTALL, Rowlands, St. Mary Cray, Kent.
- 1847 SMITH, WILLIAM J., M.D., Consulting Physician to the Weymouth Infirmary; Greenhill, Weymouth, Dorsetshire.
- 1873 SMITH, W. JOHNSON, Surgeon to the Seamen's Hospital, Greenwich.
- 1874 *SMITH, WILLIAM ROBERT, M.B., [Royal County Hospital, Winchester], 13, Crescent road, Plumstead, Kent.
- 1868 SOLLY, SAMUEL EDWIN, Colorado Springs, Colorado, U.S.
- 1865 SOUTHEY, REGINALD, M.D., Physician to, and Lecturer on Forensic Medicine at, St. Bartholomew's Hospital; 6, Harley street, Cavendish square.
- 1844 SPACKMAN, FREDERICK R., M.D., Harpenden, St. Alban's.
- 1874 SPARKS, EDWARD ISAAC, M.B. [Abroad.] *Trans.* 1.

Elected

- 1851 SPITTA, ROBERT JOHN, M.D. Lond., Clapham Common, Surrey. *Trans.* 1.
- 1875 SPITTA, EDMUND J., late Demonstrator of Anatomy at St. George's Hospital; Ivy House, Clapham Common, Surrey.
- 1843 *SPRANGER, STEPHEN, Cape Town, South Africa.
- 1854 STEVENS, HENRY, M.D., Inspector, Medical Department, Local Government Board; Greenford House, Sutton, Surrey.
- 1842 †STEWART, ALEXANDER PATRICK, M.D., Consulting Physician to the Middlesex Hospital; 75, Grosvenor street. C. 1856-7. L. 1863-8. V.P. 1871-2.
- 1859 STEWART, WILLIAM EDWARD, 16, Harley street, Cavendish square.
- 1856 STOCKER, ALONZO HENRY, M.D., Peckham House, Peckham.
- 1865 STOKES, WILLIAM, Jun., M.D., Professor of Surgery, Royal College of Surgeons, Ireland, and Surgeon to the Richmond Surgical Hospital; 3, Clare street, Merrion square, Dublin, *Trans.* 1.
- 1843 STORKS, ROBERT REEVE, Paris.
- 1858 †STREATFEILD, JOHN FREMLYN, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Ophthalmic Surgeon to University College Hospital; 15, Upper Brook street, Grosvenor square. C. 1874-5.
- 1876 STRETTON, WILLIAM HARRIS, M.D., Physician to the Farringdon Dispensary; 8, Suffolk place, Pall Mall East.
- 1871 STRONG, HENRY JOHN, M.D., 64, North End, Croydon.
- 1863 STURGES, OCTAVIUS, M.D., Assistant-Physician to, and Joint Lecturer on Medicine at, the Westminster Hospital; Assistant-Physician to the Hospital for Sick Children; 85, Wimpole street, Cavendish square.
- 1871 SUTHERLAND, HENRY, M.D., Lecturer on Insanity at the Westminster Hospital; 6, Richmond terrace, Whitehall.

Elected

- 1869 SUTRO, SIGISMUND, M.D., Senior Physician to the German Hospital; 37A, Finsbury square.
- 1871 SUTTON, HENRY GAWEN, M.B., Physician to, and Lecturer on Medicine at, the London Hospital, and Physician to the City of London Hospital for Diseases of the Chest; 9, Finsbury square. *Trans.* 1.
- 1855 SUTTON, JOHN MAULE, M.D., Medical Officer of Health; Town Hall, Oldham.
- 1861 *SWEETING, GEORGE BACON, King's Lynn, Norfolk.
- 1854 *SYMONDS, FREDERICK, Surgeon to the Radcliffe Infirmary, and Consulting Surgeon to the Oxford Dispensary; 35, Beaumont street, Oxford.
- 1870 TAIT, LAWSON, Surgeon to the Birmingham and Midland Hospital for Women; 7, Great Charles street, Birmingham. *Trans.* 1.
- 1864 TAUSSIG, GABRIEL, M.D., 70, Piazza Barberini, Rome.
- 1875 TAY, WARREN, Surgeon to the London Hospital and Surgeon to the North Eastern Hospital for Children and the Hospital for Skin Diseases, Blackfriars; 4, Finsbury square.
- 1873 TAYLOR, FREDERICK, M.D., Assistant-Physician to Gny's Hospital; 15, St. Thomas's street, Southwark.
- 1852 TAYLOR, ROBERT, 7, Lower Seymour street, Portman square.
- 1845 †TAYLOR, THOMAS, Warwick House, 1, Warwick place, Grove End road, St. John's wood.
- 1859 TEGART, EDWARD, 49, Jermyn street, St. James's.
- 1874 THIN, GEORGE, M.D., 22, Queen Anne street, Cavendish square. *Trans.* 2.
- 1862 THOMPSON, EDMUND SYMES, M.D., Physician to the Hospital for Consumption, Brompton; Gresham Professor of Medicine; 3, Upper George street, Bryanston square. S. 1871-4. *Trans.* 1. *Sci. Com.*
- 1857 THOMPSON, HENRY, M.D., Physician to the Middlesex Hospital; 53, Queen Anne street, Cavendish square.

Elected

- 1852 THOMPSON, SIR HENRY, Surgeon-Extraordinary to H.M. the King of the Belgians; Emeritus Professor of Clinical Surgery in University College, London; 35, Wimpole street, Cavendish square. C. 1869. *Trans.* 4.
- 1862 THOMPSON, REGINALD EDWARD, M.D., Assistant-Physician to the Hospital for Consumption, Brompton; 9, Cranley place, South Kensington. *Trans.* 1. *Sci. Com.*
- 1876 THORNTON, JOHN KNOWSLEY, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children; 83, Park street, Grosvenor square. *Trans.* 1.
- 1875 TIBBITS, HERBERT, F.R.C.P. Ed., Medical Superintendent of the National Hospital for the Paralysed and Epileptic; 30, New Cavendish street.
- 1848 TILT, EDWARD JOHN, M.D., Consulting Physician to the Farringdon General Dispensary and Lying-in Charity; 27, Seymour street, Portman square.
- 1872 TOMES, CHARLES S., B.A., Lecturer on Anatomy and Physiology at the Dental Hospital; 37, Cavendish square.
- 1867 TONGE, MORRIS, M.D., Harrow-on-the-Hill, Middlesex.
- 1871 *TREND, THEOPHILUS W., M.R.C.P. Edinb., Raeberry Lodge, Southampton.
- 1867 TROTTER, JOHN WILLIAM, Surgeon-Major, Coldstream Guards; Bossall Vicarage, York.
- 1859 TRUMAN, EDWIN THOMAS, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.
- 1864 TUFNELL, THOMAS JOLLIFFE, Consulting Surgeon to the City of Dublin Hospital; 58, Lower Mount street, Merrion square, Dublin. *Trans.* 1.
- 1862 TUKE, THOMAS HARRINGTON, M.D., Manor House, Chiswick, and 37, Albemarle street, Piccadilly.
- 1875 TURNER, FRANCIS CHARLEWOOD, M.A., M.D., Assistant-Physician to the London Hospital; 15, Finsbury square.
- 1873 TURNER, GEORGE BROWN, M.D., 3, Warrior square, St. Leonard's-on-Sea.
- 1876 VENN, ALBERT J., M.D., C.M., Obstetric Physician to the Metropolitan Free Hospital; Assistant Physician to the Victoria Hospital for Children; 40, Brook street, Grosvenor square.

Elected

- 1870 VENNING, EDGCOMBE, Surgeon, 1st Life Guards; Knightsbridge Barracks, and 87, Sloane street.
- 1865 VERNON, BOWATER JOHN, Ophthalmic Surgeon to St. Bartholomew's Hospital and to the West London Hospital; 43, Weymouth street, Portland place.
- 1867 VINTRAS, ACHILLE, M.D., Physician to the French Embassy and to the French Hospital, Lisle street, Leicester square; 141, Regent street.
- 1828 VULPES, BENEDETTO, M.D., Physician to the Hospital of Aversa, and the Hospital of Incurables, Naples.
- 1854 WADDINGTON, EDWARD, Auckland, New Zealand.
- 1870 WADHAM, WILLIAM, M.D., Physician to, and Lecturer on Medical Jurisprudence at, St. George's Hospital; 14, Park lane.
- 1864 WAITE, CHARLES DERBY, M.B., Senior Physician to the Westminster General Dispensary; 3, Old Burlington street.
- 1868 *WALKER, ROBERT, L.R.C.P. Edinb., Surgeon to the Carlisle Dispensary; 25, Lowther street, Carlisle.
- 1867 *WALLIS, GEORGE, Corpus Buildings, Cambridge.
- 1873 WALSHAM, WILLIAM JOHNSON, C.M., Demonstrator of Anatomy and Operative Surgery at St. Bartholomew's Hospital; Surgeon to the Metropolitan Free Hospital and to the Royal Hospital for Diseases of the Chest, City Road; 27, Weymouth street, Portland place.
- 1852 WALSHE, WALTER HAYLE, M.D., Emeritus Professor of the Principles and Practice of Medicine, University College, London; Consulting Physician to the Hospital for Consumption; 37, Queen Anne street, Cavendish square. C. 1872. *Trans.* 1.
- 1851 WALTON, HAYNES, Surgeon to St. Mary's Hospital, and to the Ophthalmic Department; 1, Brook street, Grosvenor square. *Trans.* 1. *Pro.* 1.
- 1852 WANE, DANIEL, M.D., 20, Grafton street, Berkeley square.
- 1821 WARD, WILLIAM TILLEARD, Tilleards, Stanhope, Canada.
- 1858 WARDELL, JOHN RICHARD, M.D., Calverley park, Tunbridge Wells.

Elected

- 1846 WARE, JAMES THOMAS, Tilford House, near Farnham, Surrey.
- 1818 WARE, JOHN, Clifton Down, near Bristol.
- 1866 WARING, EDWARD JOHN, M.D., 49, Clifton gardens, Maidavale.
- 1877 WARNER, FRANCIS, M.D., 15, Finsbury square.
- 1861 WATERS, A. T. HOUGHTON, M.D., Physician to the Royal Infirmary, and Lecturer on the Principles and Practice of Medicine, in the Liverpool Royal Infirmary School of Medicine; 69, Bedford street, Liverpool. *Trans.* 3.
- 1837 †WATSON, SIR THOMAS, Bart., M.D., D.C.L., F.R.S., Physician-in-Ordinary to H.M. the Queen; Consulting Physician to King's College Hospital; 16, Henrietta street, Cavendish square. C. 1840-1, 1852. V.P. 1845-6.
- 1861 †WATSON, WILLIAM SPENCER, M.B., Surgeon to the Great Northern Hospital; Surgeon to the Royal South London Ophthalmic and to the Central London Ophthalmic Hospitals; 7, Henrietta street, Cavendish square. *Trans.* 1.
- 1854 WEBB, WILLIAM, M.D., Gilkin View House, Wirksworth, Derbyshire.
- 1840 WEBB, WILLIAM WOODHAM, M.D.
- 1842 †WEBER, FREDERIC, M.D., 44, Green street, Park lane. C. 1857. V.P. 1865.
- 1857 WEBER, HERMANN, M.D., Physician to the German Hospital; 10, Grosvenor street, Grosvenor square. C. 1874-5. *Trans.* 6.
- 1844 †WEGG, WILLIAM, M.D., *Treasurer*, 15, Hertford street, Mayfair. L. 1854-8. C. 1861-2. T. 1873-7.
- 1874 WELLS, HARRY, M.D., British Vice-Consulate, Gualaguaychu, Entre Rios, Argentine Confederation.
- 1861 WELLS, JOHN SOELBERG, Professor of Ophthalmology in King's College, London, and Ophthalmic Surgeon to King's College Hospital; Surgeon to the Royal London Ophthalmic Hospital; 16, Savile row. C. 1877.

Elected

- 1854 WELLS, THOMAS SPENCER, Surgeon-in-Ordinary to H.M.'s Household; Surgeon to the Samaritan Free Hospital for Women and Children; Professor of Surgery and Pathology at the Royal College of Surgeons; 3, Upper Grosvenor street. C. 1870. *Trans.* 10. *Pro.* 1.
- 1842 †WEST, CHARLES, M.D., *President*, Corresponding Member of the Academy of Medicine of Paris; 61, Wimpole street, Cavendish square. C. 1855-6. V.P. 1863. P. 1877. *Trans.* 2. *Sci. Com.*
- 1828 WHATLEY, JOHN, M.D.
- 1875 WHIPHAM, THOMAS TILLYER, M.B., Physician to, and Lecturer on Botany at, St. George's Hospital; 37, Green street, Grosvenor square.
- 1849 WHITE, JOHN.
- 1852 WIBLIN, JOHN, M.D., Medical Inspector of Emigrants and Recruits; Southampton. *Trans.* 1.
- 1844 †WILDBORE, FREDERIC, 245, Hackney road.
- 1870 *WILKIN, JOHN F., M.D. and M.C., New Beckenham, Kent.
- 1837 WILKS, GEORGE AUGUSTUS FREDERICK, M.D., Stanbury, Torquay.
- 1863 WILKS, SAMUEL, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; 77, Grosvenor street, Grosvenor square.
- 1863 WILLETT, ALFRED, Assistant-Surgeon to, and Demonstrator of Practical Surgery at, St. Bartholomew's Hospital; Surgeon to St. Luke's Hospital; 36, Wimpole street, Cavendish square.
- 1864 WILLETT, EDMUND SPARSHALL, M.D., Resident Physician, Wyke House, Isleworth, Middlesex.
- 1840 †WILLIAMS, CHARLES JAMES BLASIUS, M.D., F.R.S., Physician-Extraordinary to H.M. the Queen; Consulting Physician to the Hospital for Consumption, Brompton [47, Upper Brook street, Grosvenor square]. C. 1849-50. V.P. 1860-1. P. 1873-4. *Trans.* 1. *Sci. Com.*
- 1859 *WILLIAMS, CHARLES, Assistant-Surgeon to the Norfolk and Norwich Hospital; 9, Prince of Wales road, Norwich.
- 1866 WILLIAMS, CHARLES THEODORE, M.D., Physician to the Hospital for Consumption, Brompton; 47, Upper Brook street, Grosvenor square. *Trans.* 3.

Elected

- 1872 WILLIAMS, JOHN, M.D., Assistant Obstetric Physician to University College Hospital; 28, Harley street, Cavendish square.
- 1859 WILLIAMS, JOSEPH, M.D. Holmhurst, Cambridge park, Twickenham.
- 1868 WILLIAMS, WILLIAM RHYS, M.D., Lecturer on Mental Diseases at St. Thomas's Hospital; Bethlehem Royal Hospital, Lambeth road.
- 1829 WILLIS, ROBERT, M.D., Barnes, Surrey. L. 1838-41.
- 1839 †WILSON, ERASMUS, F.R.S., Professor of Dermatology, Royal College of Surgeons of England; 17, Henrietta street, Cavendish square. C. 1877. *Trans.* 2.
- 1863 WILSON, ROBERT JAMES, F.R.C.P. Edin., 7, Warrior square, St. Leonard's-on-Sea, Sussex.
- 1850 *WISE, ROBERT STANTON, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Banbury, Oxfordshire.
- 1825 WISE, THOMAS ALEXANDER, M.D., Thornton, Beulah hill, Upper Norwood, Surrey.
- 1841 WOOD, GEORGE LEIGHTON, 28, Green park, Bath.
- 1851 WOOD, JOHN, F.R.S., *Vice-President*, Senior Surgeon to King's College Hospital, and Professor of Clinical Surgery in King's College, London; 68, Wimpole street. C. 1867-8. V.P. 1877. *Trans.* 3.
- 1872 WOOD, SAMUEL, St. Mary's Court, Shrewsbury.
- 1848 WOOD, WILLIAM, M.D., *Vice-President*, Physician to St. Luke's Hospital for Lunatics; 99, Harley street, Cavendish square. C. 1867-8. V.P. 1877.
- 1842 WORTHINGTON, WILLIAM COLLINS, London road, Lowestoft, Suffolk. *Trans.* 3.
- 1865 WOTTON, HENRY, 62, Bedford gardens, Kensington.

[It is particularly requested that any change of Title, Appointment, or Residence, may be communicated to the Secretaries before the 1st of October in each year, in order that the List may be made as correct as possible.]

HONORARY FELLOWS.

(Limited to Twelve.)

Elected

- 1853 BRODIE, SIR BENJAMIN COLLINS, Bart., M.A., D.C.L., F.R.S., Brockham Warren, Reigate.
- 1847 CHADWICK, EDWIN, C.B., Corresponding Member of the Academy of Moral and Political Sciences of the Institute of France; Park Cottage, East Sheen.
- 1873 CHRISTISON, SIR ROBERT, Bart., M.D., D.C.L., LL.D., Physician-in-Ordinary to H M. the Queen in Scotland; 40, Moray place, Edinburgh.
- 1868 DARWIN, CHARLES, M.A., F.R.S., Corresponding Member of the Academies of Sciences of Berlin, Stockholm, Dresden, &c.; Down, Bromley, Kent.
- 1857 FARR, WILLIAM, M.D., D.C.L., F.R.S., General Register Office, Somerset House, and Southlands, Bickley, Kent.
- 1868 HOOKER, SIR JOSEPH DALTON, M.D., C.B., K.C.S.I., D.C.L., LL.D., F.R.S., Member of the Senate of the University of London, Director of the Royal Botanic Gardens, Kew; President of the Royal Society; Corresponding Member of the Academy of Sciences of the Institute of France; Royal Gardens, Kew.
- 1868 HUXLEY, THOMAS HENRY, LL.D., F.R.S., Professor of Natural History in the Royal School of Mines; Secretary to the Royal Society; Corresponding Member of the Academies of Sciences of St. Petersburg, Berlin, Dresden, &c.; 4, Marlborough place, St. John's wood.

Elected

- 1847 OWEN, RICHARD, C.B., D.C.L., LL.D., F.R.S., Superintendent of the Natural History Departments in the British Museum ; Foreign Associate of the Academy of Sciences of the Institute of France ; Sheen Lodge, Mortlake.
- 1873 STOKES, GEORGE GABRIEL, M.A., D.C.L., LL.D., Lucasian Professor of Mathematics in the University of Cambridge ; Secretary to the Royal Society, &c. ; Lensfield Cottage, Cambridge.
- 1875 STOKES, WILLIAM, M.D., D.C.L., LL.D., F.R.S., Regius Professor of Physic at Dublin University ; 5, Merrion square north, Dublin.
- 1868 TYNDALL, JOHN, LL.D., F.R.S., Professor of Natural Philosophy in the Royal Institution ; Corresponding Member of the Academies and Societies of Sciences of Göttingen, Haarlem, Geneva, &c. ; Royal Institution, Albemarle street, Piccadilly.

FOREIGN HONORARY FELLOWS.

(Limited to Twenty.)

Elected

- 1872 BERNARD, CLAUDE, Member of the Institute of France, and of the Academy of Medicine ; Professor of Medicine at the College of France ; Professor of General Physiology at the Museum of Natural History ; Rue de Luxembourg, 24, Paris.
- 1876 BILLROTH, THEODOR, M.D., Professor of Surgery in the University of Vienna ; Vienna.
- 1864 DONDEES, FRANZ CORNELIUS, M.D., Professor of Physiology and Ophthalmology at the University of Utrecht.
- 1875 DRAPER, JOHN WILLIAM, M.A., LL.D., Emeritus Professor of Chemistry and Physiology in the University of New York ; 13, University Buildings, Washington square, New York.
- 1876 EDWARDS, H. MILNE, M.D., Member of the Institute of France, and of the Academy of Medicine ; Dean of the Faculty of Sciences and Professor at the Museum of Natural History of Paris ; 57, Rue Cuvier, Paris.
- 1835 EKSTRÖMER, CARL JOHAN, M.D., C.M., K.P.S., and W., Physician to the King of Sweden ; President of the College of Health, and Director-General of Hospitals ; Stockholm.
- 1868 GROSS, SAMUEL D., M.D., F.C.P. Philad., D.C.L. Oxon., LL.D., Professor of Surgery in the Jefferson Medical College of Philadelphia.
- 1866 HANNOVER, ADOLPH, M.D., Professor at Copenhagen.
- 1873 HELMHOLTZ, HERMANN LUDWIG FERDINAND, Professor of Physics and Physiological Optics ; Berlin.
- 1859 HENLE, J., M.D., Professor of Anatomy at Göttingen.

Elected

- 1873 HOFMANN, A. W., LL.D., Ph.D., Professor of Chemistry, Berlin.
- 1868 KÖLLIKER, ALBERT, Professor of Anatomy at Würzburg.
- 1856 LANGENBECK, BERNHARD, M.D., Professor of Surgery in the University of Berlin.
- 1868 LARREY, HIPPOLYTE BARON, Member of the Institute; Inspector of the "Service de Santé Militaire," and Member of the "Conseil de Santé des Armées;" Commander of the Legion of Honour, &c. ; Rue de Lille, 91, Paris.
- 1862 PIROGOFF, NIKOLAUS, M.D., Professor of Surgery to the Medico-Chirurgical Academy in St. Petersburg, and Director of the Anatomical Institute; Consulting Physician to the Hospitals Obuchow, Peter-Paul, and Maria Magdalena; St. Petersburg.
- 1850 ROKITANSKY, CARL FREIHERR VON, M.D., Curator of the Imperial Pathological Museum, and Professor of Pathological Anatomy at the University of Vienna. Referee for Medical and University Education to the Austrian Ministry; Vienna.
- 1856 VIRCHOW, RUDOLPH, M.D., Professor of Pathological Anatomy in the University of Berlin; Corresponding Member of the Academy of Sciences of the Institute of France; Berlin.

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SUBCUTANEOUS DIVISION OF THE NECK OF THE FEMUR

FOR

ANCHYLOSIS OF THE HIP-JOINT.

WITH A TABLE OF TWENTY-TWO CASES WHICH HAVE
BEEN OPERATED UPON UP TO THE PRESENT TIME.

BY

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Received May 9th—Read October 10th, 1876.

THE object of the present paper is to bring before this Society the results, as far as I have been enabled to obtain them, of the cases of ankylosis of the hip-joint, which have been operated upon by the subcutaneous division of the neck of the thigh bone, an operation which was first performed by myself at the Great Northern Hospital on 1st December, 1869.

During the six and a half years which have now elapsed the operation has been successfully performed in so many cases by the leading provincial and metropolitan surgeons in England, and also by Dr. Sands, of New York, and the unfavorable cases have been so few, that the subcutaneous section of the neck of the femur may

now be considered to be an established operation in surgery.

When the subcutaneous method is applied in the performance of operations of unusual magnitude, such as division of the neck of the thigh-bone, division of the shaft of the femur or humerus, it would be unreasonable to expect the uniformly favorable results which follow the subcutaneous division of tendons, because the condition upon which the safety of the subcutaneous method depends cannot all with certainty be fulfilled. In these large operations the air may be effectually excluded, but the injury to the deeply seated structures may be excessive, either from cutting or laceration by the saw. Hence the absolute immunity from inflammation or suppuration claimed for the subcutaneous method is not maintained, and deep-seated suppuration will occasionally follow such operations, although the rarity of such a result has astonished every surgeon who has practised them.

In a very large proportion of the cases operated upon, subcutaneous division of the neck of the thigh-bone has proved to be as harmless an operation and as free from all risks as subcutaneous tenotomy; and there can be no doubt that such success largely depends upon the attention paid to the following points—first, the selection of appropriate cases; and, secondly, the care taken by the operator in performing the operation strictly in accordance with the laws of subcutaneous surgery as to the exclusion of air, and the smallest possible disturbance or laceration by the saw of the tissues surrounding the bone.

Nevertheless, as the cases operated upon have multiplied in number, there has been a certain proportion of bad results, such as local suppuration, with its more serious complication, pyæmia, leading to the death of the patient. I am not aware, however, that this result has followed in more than one case out of the total number of twenty-two operated upon up to the present time. This occurred in a boy, aged eight years, who had fibrous ankylosis of the left hip, with the limb in a deformed position, and was

operated upon by Mr. Croft at St. Thomas' Hospital March 4th, 1871. There had been abscesses in the neighbourhood of the joint some years previously. Attempts at forcible extension had been employed a week before the operation, when the contracted muscles were also divided, so that perhaps the articulation was not in a sufficiently quiescent state at the time of the operation. Suppuration followed, and the child died of pyæmia on March 30th, 1871.*

A second death, however, in a girl, aged eighteen, not quickly following the operation, from pyæmia, but occurring eight months afterwards, in consequence of prolonged suppuration, followed by symptoms of kidney disease, with albuminuria, and also tubercular deposit in the lungs, may be added to the list, making a total of two deaths.

This case, like Mr. Croft's, belonged to the most unfavorable class, viz. advanced strumous disease with abscess, the fistulous openings from which were indicated by two depressed cicatrices. It belonged to the class which I had decided to reject, but it presented some favorable points, and, emboldened by previous success, I departed from my own law, and have reason to regret so doing.

This was the last of the five cases in which I have performed the operation. All the others terminated favorably, although in one a deep abscess formed six weeks after the operation, the wound of which had healed soundly without suppuration. In the other three cases no suppuration whatever occurred, and they proceeded with as little local disturbance as in ordinary cases of tenotomy.

Such a result bears good testimony to the general safety of the operation, and although the dangers of all operations must increase with their magnitude, these may be kept within the narrowest possible limits by the

* This case is described in a pamphlet 'A New Operation for Bony Anchylosis of the Hip-joint with Malposition of the Limb by Subcutaneous Division of the Neck of the Thigh-bone,' by Wm. Adams, 1871, p. 19. London: J. and A. Churchill.

judgment of the surgeon in selecting his cases, and the skill shown in the performance of the operation.

Amongst recent surgical writers, Mr. Holmes is the only one who speaks of the operation with distrust, and refers to the difficulty of performing it in a truly subcutaneous manner. Mr. Holmes observes,* “ But in cases which really require any such operation, *i. e.* where the changes in the relative position of the bones is considerable, there is often a very large deposit of bone around the joint, and the shape and size of the anchylosed articular ends has been much altered. It may, therefore, be impossible in such cases to execute any section really deserving of the title subcutaneous, and the operation approaches in gravity and in extent to that of excision, and is liable, like excision, to be followed by renewed disease in the divided surfaces by exhausting suppuration, or by pyæmia.”

To this I would reply that in any particular case of bony ankylosis of the hip-joint, when there is a doubt as to the possibility of the neck of the femur being divided subcutaneously, in consequence of alterations which may have taken place at the articulation, such as described by Mr. Holmes, it should not be attempted; but some other operation below the joint, such as that first proposed and successfully performed by Mr. Gant, should be adopted.

Mr. Gant has, in two cases of ankylosis of the hip-joint with malposition of the limb, successfully divided the shaft of the femur subcutaneously, just below the small trochanter, using instruments similar to those employed by myself; but the saw had a longer cutting edge, and a thinner blade—the width the same.

The following account of Mr. Gant's first operation is taken from the ‘Lancet’ December 21st, 1872. “Ankylosis of the hip-joint, with extreme malposition of the limb upwards and inwards; subcutaneous section of the femur below both trochanters.

* ‘Treatise on Surgery, its Principles and Practice,’ by T. Holmes, M.A. Cantab. Smith, Elder, and Co., 1875.

“On December 10th Mr. Gant performed the above operation at the Royal Free Hospital on a boy, aged six years, remarking that it differed both from Mr. Adams’ subcutaneous section through the neck of the femur, and from the American operations of section and incision between the trochanters, by Barton and Sayre.

“The reasons which induced Mr. Gant to perform this new operation had a double foundation. First, on the pathological ground that the section is not made in a diseased portion of the femur, but in sound bone. Second, the anatomical reason that, the section being made below the small trochanter, the resisting psoas and iliacus muscles are set free. Further, that there is no risk of atrophy or necrosis of the head of the bone from cutting off its vascular supply.

“Mr. Gant thinks that this operation will be applicable, instead of excision, to a class of hip-joint cases where the disease is quiescent, but the limb useless, the state being simply that of ankylosis with malposition, and especially appropriate in young subjects where removal of the epiphysal head of the femur might be followed by further shortening of the limb.

“After section the limb was at once freely movable, and being brought down into a straight position it was fixed by means of sand bags; extension was maintained by a weight from the foot, and counter extension by a perineal band.

“*Result.*—Primary union of the wound; osseous union of the femur; an angle can be felt at the junction of the section ends of the bone, but this slight deformity is not visible externally; the limb is perfectly straight, and the boy can stand and walk without any inconvenience or limp.”

Mr. Gant has since performed this operation in another case, a boy æt. 14. Firm union took place, but the permanent result in regard to the use of the limb is, he informs me, unknown to him. In Mr. Gant’s operation the true subcutaneous character is preserved, together with

its immunity from inflammation and suppuration, hence its success.

I cannot agree with Mr. Gant in the reasons he has assigned for preferring this operation to my own, or even as to the class of cases to which he considers it applicable, but I regard it as admirably adapted to the class of cases indicated by Mr. Holmes, viz. those attended with considerable alterations in the joint, and often with new bone thrown out in its neighbourhood, in which my operation would be liable to fail, or be followed by deep suppuration.

Mr. Gant's operation has also the merit of being much easier of performance, as the bone is reached immediately under the skin; and if the point of the saw be kept very close to the bone, there need be very little disturbance of the surrounding tissues, or risk of hæmorrhage.

I have twice divided, with the subcutaneous saw, the shaft of the femur for great deformity resulting from badly-united fractures, and once the shaft of the humerus for straight ankylosis. No suppuration whatever occurred in these cases, and the operations were performed without difficulty.

Mr. Maunder divided the neck of the femur subcutaneously, in the manner I have recommended, in a case of fibrous ankylosis in a child eight years of age on 1st July, 1874. See 'Lancet,' July 11th, 1874. The child was the subject of dislocation of the left femur on the dorsum ilii, consequent upon hip-joint disease. No suppuration occurred.

Mr. Maunder has also performed operations on the shaft of the femur in a subcutaneous manner in cases of deformity. In a recent number of the 'Lancet,' March 25th, 1876, it is stated, "Lately, at the London Hospital, Mr. Maunder has practised section of bone with chisel and mallet in three instances. In each case the femur was the bone divided. In one case (that of an adult) slight suppuration occurred, while in two (a child and an adult) primary union took place. The shaft of the bone was

divided to remedy distressing deformity, and the patients are now in a position to to become useful members of, rather than remain burdens on, society."

Imperfectly subcutaneous operations, such as those here referred to, are applicable only to certain cases, and are essentially the same as those performed by Langenbeck, Meyer, Pancoast of Philadelphia, Brainard, L. S. Little, and others. Such operations, although successful in many instances, must be attended with a much larger percentage of unfavorable results than the true subcutaneous operations, and are not applicable to the hip-joint from the dangers of deep suppuration which must attend such a procedure.

Mr. Bryant, in the second edition of his 'Practice of Surgery,' just published, speaks very favorably of the operation proposed by myself, and in dividing the neck of the bone subcutaneously, adopted precisely the method I have recommended.

Mr. Bryant observes, "I have performed the operation with marked success in three cases. . . . In each of my own cases the neck of the thigh-bone was readily divided in less than five minutes, and the wound above the trochanter healed in a few days; the boys, aged eight, fifteen, and sixteen respectively, walked in eight weeks. I did not attempt to obtain movement in any of these cases, being well satisfied with the result secured."

With regard to obtaining useful motion and a false joint as the result of the operation, I may state that I have never succeeded in so doing. I did not attempt it in my first case, but after the success obtained by Mr. Jessop, of Leeds, in which he obtained useful motion in two cases, I tried it in two cases with some success for a short time. Although I could obtain some motion under chloroform, it was quickly lost, partly from the inclination to stiffen, as well as the unwillingness of the patients to submit to chloroform, and subsequent pain in the attempts to preserve motion by the passive movements adopted. I

therefore abandoned the attempt, and was satisfied in obtaining ankylosis in the straight position.

A most remarkable and successful case, however, in which useful motion has been obtained in both hip-joints, after subcutaneous division of the neck of the femur for ankylosis, with both legs in a straight line with the body, has been published by Mr. Lund, of Manchester.*

In the case operated upon by Dr. Sands, of New York, useful motion was obtained, and existed eight months after the operation, when the patient was exhibited to the New York County Medical Society, October 27th, 1872. In this case Dr. Sands observes, "After the operation the patient was confined to bed for six weeks, in the hope of obtaining bony ankylosis in the straight positions; but as it was found at the end of that time that the parts remained freely moveable he was permitted to get up and to move about the ward on crutches.†

In Mr. Sydney Jones' remarkable case of bony ankylosis of both hip-joints, with the thighs in an extremely abducted position, the operation was successfully performed on both hips, and no suppuration occurred. In this case tolerably free motion was kept up for some time, and under chloroform the motion was very free, but some inclination to stiffen was apparent when I last saw it.

Before concluding this paper I would mention, as a remarkable circumstance, that Mr., afterwards Sir C. Bell, proposed the operation of dividing the neck of the thigh-bone in a manner approaching to the true subcutaneous method, not for bony ankylosis of the joint with deformity, for which my operation was designed, but he proposed that it should be performed during the progress of the disease for the purpose of allowing the head of the bone to remain at rest in the acetabulum—in fact, merely to

* 'Brit. Med. Journ.,' May 29th, 1876.

† "A Case of Bony Ankylosis of Hip-joint successfully treated by Subcutaneous Division of the Neck of the Femur," by H. B. Sands, M.D., Surgeon to the Bellevue and Roosevelt Hospitals (reprinted from the 'New York Med. Journ.,' December, 1873), New York. Appleton and Co., 1873.

secure immobility—believing that this would contribute in a great degree to the production of bony ankylosis, and that a freely movable joint would remain at the seat of operation, so that the effects of ankylosis, as to the loss of motion and the production of deformity, would be prevented.

In reference to the operation Sir C. Bell observes, “Such a cut as would permit the use of a small saw to divide the neck of the bone would not be a formidable operation.”*

Had this operation, however, been performed during the progress of the disease, I believe its author would have been disappointed, at least in the majority of cases, as to the production of false joint, as the patients could not have borne the rough manipulation by the frequent repetition of flexion, extension, and rotation necessary to maintain free motion. Still, the operation proposed by Sir Charles Bell is of such a remarkable character, and of so much interest in connection with that proposed by myself, which, to a certain extent, it foreshadowed, that I thought it due to this illustrious surgeon that it should be noticed in the present paper. It is the more remarkable as having been published in the year 1828, three years before Stromeyer’s operation of subcutaneous tenotomy, from which the introduction of subcutaneous surgery may be dated.

This proposed operation of Sir C. Bell was first brought under my notice by my colleague, Mr. Carr Jackson, long after the publication of my paper.

* The ‘London Medical Gazette,’ January 12th, 1828, p. 139.

Note to last case in Table on p. 10:—Mr. Willett has since communicated, with reference to this case, that deep suppuration, followed by necrosis, ensued. Exarticulation at the hip was performed on February 26th, 1877, and the patient died the same day.

Table of 22 cases in which the subcutaneous division of the neck of the femur has been performed.

| Date. | Surgeon. | Termination. | One or both hips. |
|-----------------------------------|--|---|-------------------|
| December 1, 1869 | W. Adams, Great Northern Hospital | Successful | One |
| August 25, 1870 | T. R. Jessop, Leeds | " | " |
| November, 1870 | F. Jordan, Birmingham | " | " |
| December 5, 1870 | F. W. Jowers, Brighton | " | " |
| March 4, 1871 | J. Croft, St. Thomas's Hospital | Fatal from pyæmia | " |
| March 16, 1871 | T. R. Jessop, Leeds | Successful | " |
| June 7, 1871 | J. Hardie, Manchester | " | " |
| November 1, 1871 | W. Adams, Great Northern Hospital | " | " |
| January 24, 1872 | W. Adams, Great Northern Hospital | " | " |
| February 12, 1872 | Dr. Sands, New York | " | " |
| April 24, 1872 | W. Adams, Great Northern Hospital | " | " |
| 1874 | T. Bryant, Guy's Hospital | " | " |
| 1874 | T. Bryant, Guy's Hospital | " | " |
| July 1, 1874 | C. F. Maunder, London Hospital | " | " |
| July 8, 1874 | W. Adams, Great Northern Hospital. | Fatal 8 months after operation from suppurati- on and kidney disease | " |
| March 5, 1875 } May 28, 1875 } | E. Lund, Manchester | Successful | Both |
| June 2, 1875 } | Sydney Jones, St. Thomas's Hospital | " | " |
| October 13, 1875 } | T. Bryant, Guy's Hospital | " | One |
| November 21, 1875 } | J. Hutchinson, London Hospital | " | " |
| November 1875 } | A. Willett, St. Bartholomew's Hospital | Under treatment | " |
| *January 1876 | * See note on previous page. | | 22 operations |

A CASE
OF
RESECTION OF THE TARSAL BONES
FOR
DOUBLE CONGENITAL TALIPES
EQUINO-VARUS.

BY
J. N. C. DAVIES-COLLEY, F.R.C.S.,
ASSISTANT-SURGEON TO, AND LECTURER ON ANATOMY AT, GUY'S HOSPITAL.

Received October 6th—Read October 10th, 1876.

ALTHOUGH great progress has been made of late years in the cure of talipes by the introduction of subcutaneous tenotomy, and the improvement of mechanical appliances, there will always remain a number of cases, which, from the age to which they have attained, the rigidity of the tissues, and the altered shape of the bones, present insurmountable obstacles to treatment by the ordinary methods. It is not improbable that many of these cases might be successfully treated in the way which was adopted in the following instance :

Edwin H—, æt. 12, was admitted into Guy's Hospital on May 8th, 1875, under the care of Mr. Cooper Forster. From birth he had suffered from deformity of both feet. When he was four years old he was taken to Gloucester Infirmary, where some tendons were divided, but without

any beneficial result. Since then he has not been under any treatment whatever.

On admission the anterior part of each foot is turned inwards at a right angle to the rest of the foot, and is twisted in such a way that the patient walks on the outer edge of the dorsum, while the sole looks backwards. The heels are raised, especially that of the left foot. On the dorsal aspect of the external cuneiform and cuboid bones bursæ have formed, and that upon the right side is suppurating. He stands with his legs about a foot and a half apart, and he can only walk, or rather waddle, with great pain and difficulty.

He was kept in bed, and the bursal inflammation soon subsided. Splints were applied to the outer side of the legs, and an attempt was made to improve the position of the feet by elastic bands.

During Mr. Cooper Forster's absence in September, the boy came under my care. He was then in much the same condition as when he came in, except that the supuration of the bursa on the dorsum had disappeared. I found that the feet were quite rigid, and that hardly any improvement could be produced in them by the application of force. I first divided subcutaneously the tendo Achillis, the tibialis posticus tendon, and the plantar fascia of the left, which was the more deformed foot. As very little was gained by this operation and the subsequent treatment, I resolved to adopt severer measures. It was at first my intention to remove the cuboid bone. Mr. Howse, however, suggested to me that it would be far better to cut out a wedge-shaped piece of the tarsus without paying any regard to its articulations.

Acting upon this advice of my colleague, I decided to expose the bones of the instep, and then to saw it through in two places in such a way that the two sections should meet at the inner border and diverge towards the outer border of the foot. I thought, also, that it would be easy to arrange that the upper surface of the segment removed should be somewhat more extensive than the lower, and

that in this way the peculiar twist of the foot, which contracted at the same time the sole and its inner border, might be remedied.

On October 12th chloroform was administered, and after an Esmarch's bandage had been applied, I made an incision three inches in length along the outer border of the left foot, from the middle of the os calcis to the middle of the fifth metatarsal bone. From the centre of this incision I carried another two inches long transversely across part of the dorsum, dividing the tendons of the peroneus longus, the peroneus brevis, and the extensor brevis digitorum. When I had carefully reflected the soft parts, I found that I had to modify my plans. I could not expose the whole of the triangular surface, which I had intended to saw out, without dividing the extensor tendons, or making an incision upon the inner side of the foot. I therefore decided to remove first the cuboid. This was readily done by means of the scalpel and elevator, and the bone was found to be of normal size and shape. Then I sawed off the great process of the os calcis in a plane looking forwards, outwards, and upwards. Next, by means of an elevator, bone-forceps, and dressing-forceps, I took away piecemeal portions of the three cuneiform bones, for the deformity could not be reduced until I had excavated as far as the inner border of the foot. I then pared off some more of the os calcis with a strong knife, and continuing the plane of section inwards I cut away nearly all the scaphoid and a part of the head of the astragalus.

Finally, I removed the articular cartilage from the base of the two outer metatarsal bones. The foot could now be easily placed in the normal position, and the osseous surfaces fitted each other with a fair degree of accuracy. The soft parts of the sole had not been touched.

On relaxing the elastic tourniquet, a brisk hæmorrhage ensued. The wound was dressed with carbolic gauze. The antiseptic spray was used during the operation and at each dressing.

It was found necessary, on account of the continuous oozing of blood from the bones, to put in a sponge soaked in carbolic lotion, and to keep it there until a week after. Very little systemic disturbance followed the operation. His temperature only once reached 101° . I at first had some difficulty in keeping the foot in good position, but at the beginning of November I adopted a plan which enabled us to adjust the surfaces and to dress the wound very easily. It consisted of a back-splint extending from the middle of the thigh to within four inches of the heel. To the distal end of this was fastened a transverse bar of wood, terminating on either side in short upright bars, to which I attached the fore part of the foot by means of strapping.

By November 23rd the left foot was nearly healed, so I decided to operate upon the right. I at once proceeded to resect the tarsus, without any previous division of the tendons. The bones were so soft that I was able to pare them away without using the saw. In every other respect the operation was the same as that performed upon the other foot.

Again there was a troublesome oozing of blood, which could only be restrained by the retention of a sponge between the cut surfaces. He made a rapid recovery. His temperature only once rose to 102° , and was usually below 100° . There was but little discharge, and no burrowing of pus. The same apparatus was used to draw the front part of the foot into position.

By the 1st of January he was able to get about in a wheel-chair.

On February 3rd, less than ten weeks from the second operation, the wounds were quite healed, and he left off the dressings and splints. A few days before he had begun to walk with assistance. He could now walk by himself, and no apparatus was used from that time.

In March he went to the Bognor Convalescent Establishment.

Upon his return in April I examined him carefully. The feet were quite free from any tenderness. They were rather short for a boy of his age, and the heels projected somewhat backwards. I was surprised to find that the arch of each instep was as good as in a well-formed foot. The movement of the ankles was considerably improved, though not as free as it should be. He could walk well,

FIG. 1.



Before operation.

FIG. 2.



After operation.

treading upon the whole of the sole. The strength of the feet was also shown by the fact that he could jump and hop. The movement of the toes was perfect, and the peronei had acquired some firm attachment to the outer side of the foot. I could not, however, satisfy myself that the peroneus longus had any influence upon its inner border. The cicatrices of the incisions had contracted so

much that their original direction could no longer be recognised. No depression remained to show where the tarsal bones had been removed, and I doubt very much whether any one who examined the feet would have suspected that so much had been taken away. The woodcut (Fig. 2) shows the condition of the feet at this time, and as I brought him here on the 25th of last April some of the Fellows were then able to test by actual inspection the completeness of his recovery.

In September his father wrote to say that he was quite well and able to work, and that he had lately walked six miles with but little fatigue.

Remarks.—Since I operated upon this patient, I have learned from Mr. Adams' interesting work on club-foot that Dr. Little formerly suggested that "in inveterate varus, the treatment might well be commenced in robust subjects by ablation of the os cuboides." In the Society's 'Transactions' of 1857 there is an account by the late Mr. Solly of a case in which he removed all the cuboid, except the posterior articular surface, in both feet of a young gentleman, aged 23. Mechanical treatment was then used and the patient made a fair recovery. Mr. Solly considered that the operation materially expedited his cure.

Mr. Davy has recently published a series of cases in which removal of the cuboid bone had been followed by good results. I have not heard of any other instances in which this treatment has been adopted.

The chief points in the operation which Mr. Howse suggested and I performed upon this patient appears to me preferable to that just mentioned, are the following :

1. After resection the foot can be placed at once, if desired, in its proper position. Certainly in my own case the removal of the cuboid alone would not have allowed of this.

2. There is no large cavity left which has to be filled up with reparative material. Moreover, as the bony surfaces are exposed, and the articular cartilages taken away,

I consider that fibrous, and possibly bony, union is likely to be established with greater rapidity.

3. The patient will probably be able to discard the use of apparatus much sooner, for little, if any, traction has to be used in order to restore the proper shape of the foot. In the case I have related the patient began to walk without any apparatus in ten weeks after the second resection, and he has never had to wear any since, whereas the patients of Mr. Solly and Mr. Davy appear to have required to use mechanical appliances for a long time afterwards.

With respect to the operation itself and the subsequent treatment, the following points may be deduced from the single successful case which I have brought before you.

1. It is not necessary to divide the tendo Achillis before the resection. If the elevation of the heel be not great it may not be requisite to perform tenotomy at all.

2. Esmarch's bandage is of great use, as in the bloodless state of the tissues it is easy to remove such portions of bones as will leave two plane surfaces suited for exact coaptation.

3. It is better to begin by removing the cuboid, and then to cut or saw off such portions of the adjacent bones as must be taken away, in order that the foot may be placed in a good position without the employment of much force.

4. As so many synovial cavities and sheaths of tendons have to be opened, the dangers of the operation are much diminished by the careful use of antiseptic precautions.

5. The subsequent employment of such a splint as that which was used in this case facilitates very much the maintenance of the foot in good position, and renders the change of dressing a simple and almost painless proceeding. I may add that I have since used splints made upon this principle for the treatment of ordinary cases of talipes, and have found them very efficient in everting and abducting the front part of the foot.

In conclusion, I would submit that this operation may

be adopted with advantage in many cases where ordinary methods have been employed without success, or where the parts are so deformed and rigid that ordinary methods are likely to fail.

I should also recommend it in cases where it is desirable for the patient to avoid the long and painful treatment and the expensive apparatus which are required for the cure of the severer examples of this deformity by ordinary methods.

NOTES OF A CASE
OF
PSEUDO-HYPERTROPHIC PARALYSIS,
WITH A FEW OBSERVATIONS ON SURFACE
THERMOMETRY.

BY
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IN a case of the above-mentioned disease, which I had the honour of reporting to this Society three years ago, the overgrown calves appeared to be constantly warmer than the thighs. I take now the opportunity of recording a case in which the thighs were found to be warmer than the calves, and, in connection with the discrepancy, of making some remarks on methods and conditions of the taking of the temperature of the surface of the body, particularly with reference to simultaneous variations of the temperature in different parts.

William M—, a boy of 7 years, was admitted into St. Thomas's Hospital on the 30th June, 1876, under the care of Dr. Bristowe, to whose kindness I owe the advantage of being able to investigate the case.

The boy's mother stated that he had been weakly from

birth, and that for at least four years he had been getting weaker on his legs. Of late he had been in the habit of falling about, his falls being chiefly on his face. He had not been known to lose consciousness on any occasion, nor to be at any time convulsed. His father was stated to be consumptive. His mother had had chorea before marriage, and described herself as being still excessively nervous. Of four other children, all younger than the subject of the present history, one, a boy of six, was beginning to be affected with pseudo-hypertrophic paralysis; a second, a girl, died in convulsions during teething; a third, aged two years and a half, was said to be healthy; and the last, an infant, was alleged to be very weakly. There was, as far as could be ascertained, no history of syphilis in the parents, but as the father did not appear for examination, the point was not fully decided.

William M—, whose photograph is placed before the Society, is a fair-haired, somewhat ruddy child, three feet five inches in height, and in weight two stone seven pounds and a half. His expression is not exactly vacant, nor indeed dull, but a sort of apish look is produced by a habit which he has of raising the eyebrows and wrinkling the forehead. His intellectual power appears to be by no means defective. He has been to school, has learned to read, and, considering that his schooling has been much broken by his illness, he shows certainly an average capacity of acquiring knowledge. In manner he is lively and cheerful, with a disposition to impish mischief and tricks.

When stripped and examined in the erect posture he is found to stand with the feet planted about six inches apart, with the buttocks thrown back, the back bent deeply forwards, and the shoulders again thrown back beyond the line of the buttocks. He has difficulty in remaining perfectly still, tending obviously to fall forwards. The heels are raised from the ground, and the body balanced for a few seconds on the balls of the great

toes. A strong backward movement of the shoulders is necessary to reinstate him on the soles of his feet. As long as he continues to stand this backward-and-forward swaying movement goes on. He not seldom loses equilibrium, and either falls or shows a tendency to a fall, only averted by the assistance of some fixed object to which he clings. While he stands the muscles of the upper limbs twitch constantly, and he often seems to try to grasp the floor for support by strongly flexing his toes.

The muscles of the calf are enlarged distinctly out of proportion with the other muscles of the body. They are abnormally firm when at rest, and during contraction they become as hard and knotted as the biceps of a blacksmith. The right shoulder droops, and the right deltoid is enlarged and hardened, while the other muscles of the shoulder are perhaps a little wasted. The left deltoid is also larger than proportion would demand. The remarkable forward curvature of the back is due to weakness of the muscles; the vertebræ are not diseased and are free; the sacro-lumbar muscles appear to be increased in bulk. The feet are not large and the insteps are high. The following measurements of various parts of the body have been taken:

| | | | |
|---|---|---|-------------------|
| Circumference of head from occipital protuberance | | | |
| round frontal protuberance | . | . | 19 inches. |
| Circumference of chest at nipples | . | . | 22 " |
| Arm round deltoid | . | . | $6\frac{3}{4}$ " |
| Arm below insertion of pectoralis major | . | . | 6 " |
| Forearm, broadest part | . | . | $6\frac{1}{4}$ " |
| Wrist | . | . | $4\frac{1}{2}$ " |
| Thigh | . | . | $11\frac{1}{2}$ " |
| Calf | . | . | $9\frac{1}{2}$ " |
| Ankle | . | . | 6 " |

The limbs of the two sides agreed in their measurements.

Movements.—In walking he moves cringingly, like a bather going over a shingly beach, holding the shoulders in the air and putting his feet down in a hesitating way. As the bather tries to make the pressure of his feet on the

ground as light as possible, the patient evidently tries to help and sustain the weak lower limbs by the exertion of the upper. In advancing, he raises the upper arm to a horizontal position and keeps the two arms in line with each other and the shoulder, making, as Dr. Twining, House Physician at the time, suggested, a use of the arms comparable to the use of a balancing-pole. At each forward step one shoulder is thrust forward, and on this, as a pivot, the other is then swung in turn forward, dragging the pelvis after it, and so pulling the foot along the floor. These are movements made when he is naked. With boots or shoes on his feet he makes a more rapid shuffling movement, being able to slide his feet along the floor. He can make an attempt at a run, but it is only a quicker shuffle. In walking more than in standing he is apt to fall. His fall is a collapse, like that of a dead body from which a support hitherto keeping it in an erect position has been suddenly removed. It seems as though, for an instant, the nervous control of the muscles were suspended, letting the body come crashing in a heap to the ground with the face downward. His face, chest, and arms present many bruises received in these falls.

In rising from the ground he begins by raising one knee to the kneeling position with the foot well planted, and then, placing his hand on the knee, raises his body by the shoulders very painfully, swaying in an uncertain and wavering fashion which has been compared by my clinical clerk, Mr. Gulliver, to the movements of an acrobat perched upon a rope, who is exchanging under the pressure of weight the stooping for the erect position. Mr. Gulliver also observed him ascending stairs and noticed that, at each step, the body was raised by the agency of the hand placed upon the knee. The extensors of the foot must be weak, inasmuch as when he hangs a foot out of bed he cannot bring it to an acute angle, indeed, scarcely to a right angle with the leg. In the same position he cannot raise his knee towards his body unless with the aid of his hands.

The general weakness of the back and lower limbs is again shown by his movements in bed. These start from the shoulders. If he wish to turn from side to side he twists the shoulders round and drags the body after. Not always however, for unless his purchase is good the body remains helpless, and, without external aid, the change of position is not effected. In all these various directions of combined movement, no such assistance is given by the great extensors of the foot, as might have been expected if their strength had been proportioned to their bulk and hardness.

As a general result of all observations it may be decidedly concluded that there is a general failure of muscular power, and that this failure is most conspicuous in the back and legs.

Sensation.—The skin presents no unusual appearance and no variations of vascularity, except that the exposed parts are more ruddy.

The sensibility of the skin is good. In the calves the limit of confusion, when callipers are applied, is fifteen sixteenths of an inch. In the forearm it is about the same. Generally speaking discriminative sensation is a little more acute on the right side of the body than on the left.

Electrical sensibility.—The muscles of the body generally, and of the calves in particular, were less than naturally sensitive to faradic electricity. The unruliness of the patient under this kind of observation interfered, however, with the attainment of accurate results.

The constant current being used it was found that the calves did not contract at the closure and opening of the circuit with less than thirty-six cells of a Stohrer's battery, the thighs with less than thirty, and the sacro-lumbar muscles with less than twenty to twenty-four.

Microscopic examination of the muscles of the calves.—Portions of the muscles of both calves were removed for examination by means of the *emporte-pièce*, made for me by Mr. Hawksley, and described in my former paper. I

exhibit it now). On the first occasion the instrument brought away from both legs a very large proportion of adipose tissue mixed with a small quantity of muscle. Supposing that the interval between two muscles had been reached I renewed the experiment, and, though satisfied that I explored the middle of the gastrocnemius, I still brought away on two occasions a preponderance of adipose tissue.

Dr. Charles, Demonstrator of Physiology, has been good enough to make examination of the muscles removed on the various occasions, and I submit his report, which describes muscular structure of a far from natural character.

“The three pieces of muscle were prepared as follows :

“First piece was examined in glycerine after having remained several days in monochromate of ammonia 2 per cent. sol.

“Second piece was stained with hæmatoxylin sol. and examined in Dammar varnish, after having remained four days in chromic acid $\frac{1}{5}$ per cent.

“Third piece was teased and stained with hæmat. sol. almost immediately after its removal from the limb ; it was mounted in Canada balsam in the usual way.

“The following points were noticed :

“(a) That many of the fibres were very small, and that they all presented very close transverse striation.

“(β) That in some of them the striation was obscure or very badly marked, here and there often being replaced by a cloudy or finely granular appearance.

“(γ) That between many of the fibres was a coarsely granular material abundantly provided with nuclei.

“(δ) That many of the fibres appeared quite healthy, and that well-marked fatty degeneration could nowhere be detected.”

Before making any statements regarding the temperature of the limbs, I will state that the viscera of the chest and abdomen appeared to be quite healthy ; and as the question of temperature will occupy the rest of the

paper, it will be convenient to introduce here a few notes regarding the younger brother of the patient.

Henry M—, æt. 6, the next brother of the patient, was brought to me once or twice for inspection. He exhibited generally the symptoms of his brother, but in a lower degree. He could walk and run with only a little difficulty, and could stoop and raise himself without assistance. There was evident enlargement of the calves, and his gait and attitudes were a delicate caricature of his brother's.

The information to be got by testing him was limited, the child being of defective intellect.

The following measurements were taken :

| | |
|---|--------------------------|
| Circumference of head | 19 $\frac{1}{8}$ inches. |
| Circumference of chest | 19 $\frac{1}{4}$ „ |
| Right calf | 7 $\frac{7}{8}$ „ |
| Left calf | 8 $\frac{1}{8}$ „ |
| Right thigh not measured, being distorted by tumours. | |
| Left thigh | 11 „ |
| Arm, middle | 6 „ |
| Forearm, middle | 6 „ |

Temperature.—Many observations of the temperature of the calves and thighs were made, and circumstances occurring in the process of observation directed my attention to several points connected with the estimation of the temperature of the surface of the body.

It is, in the first place, obvious that too very distinct lines of observation are open—first, of absolute temperature in one part, or many; second, of relative temperature of different parts. The object which I originally had before me was to arrive at a knowledge of the relative temperature of the muscles of the overgrown calves and of the less developed thighs. It was not in my power to insert any thermometrical apparatus into the muscles, as had been done by Becquerel and Breschet in the case of a few young men, and by many observers in animals; and for the object in view it was necessary to get more than the temperature of the surface, for there was, in my mind, an idea of

the possibility of a rise of temperature in the muscles independently of the skin; and from many observations it appears pretty certain that the temperature of the skin when exposed to the air does not at all correspond with the temperature of subjacent parts. In relation to this I would refer especially to Mr. A. H. Garrod's paper on some of the minor fluctuations in the temperature of the human body, published in the 'Proceedings of the Royal Society' for 1869. Mr. Garrod here, among other interesting statements, gives a series of tables showing that on stripping the warm body of clothing in a cold air the temperature under the tongue rose at the same time that the surface became colder, and shows reason for explaining the change by local alterations of arterial tension. Again, the very careful electro-thermal observations of Becquerel and Breschet give ground for believing that the muscles are decidedly warmer than the connective tissues around them.

In comparing, therefore, the calf with the thigh I endeavoured in the first place to place them under exactly similar conditions of rest, exposure, and pressure. That is to say, the patient was placed flat on his back, not turned to either side, his legs were kept straightly extended, and he was covered with a light blanket. For reasons which I will afterwards more fully explain, I did not use thermo-electrical apparatus, but a pair of very accurate and very accurately corresponding mercurial thermometers made for me by Mr. Hawksley. They were made after the plan of some which I had seen used by our lamented friend, Dr. Sibson, in observations on the temperature of the face in thoracic aneurism. I now exhibit a pair of these which, in practice, were either supported by a boxwood backing, or were strapped on to the skin and covered with cotton wool. I exhibit also a beautiful and very perfect arrangement devised by Dr. Dupré. For my purpose Mr. Hawksley has devised and made two thermometers, having like these the bulb drawn out into a flat spiral, so as to expose a large surface on

each side, but having added to them pads and means of fixation, which make them more available for my purpose. Mr. Garrod relates that he used a flat spiral thermometer for ascertaining the temperature of the pectoral region, but does not mention that it was covered in any way on the free surface when the opposite surface was applied to the skin. Mr. Hawskley has made me a circular pad or cushion of glove-kid stuffed with cotton wool. The pad is two inches in diameter, is five eighths of an inch thick in its centre, and thins very gradually to the margins, where it is supported by a rim of wire, and has a socket to be attached to the stem of the thermometer. The stem being thrust through the socket the bulb lies flat against one side of the cushion. To the other side is sewn an elastic band with tapes attached. By this arrangement the bulb is readily bound against any surface, and when properly fixed is inclosed in a chamber with non-conducting walls, so that loss of heat from its free surface and from the free surface of the adjoining skin is prevented, and the temperature of the parts beneath the skin is allowed to influence the thermometer, if exposure be maintained for a sufficiently long period. The thermometers are non-registering and have to be read *in situ*. Within the last few weeks I have learnt from one of Dr. John Davy's earlier papers that he made use of an instrument constructed upon similar principles for the same purpose. In 1839 he wrote as follows:—"That the thermometer might be applied equally to all parts of the surface; its bulb, in form nearly cylindrical, was fixed to a small piece of cork hollowed and lined with fine wool, and thus half its superficies was applied in each instance."

With the above-described instruments, and with the observance of the conditions above laid down, eleven comparative observations were made on William Marshall:

| | | | | |
|----------------------------|---|------------------|---|--------------------|
| Aug. 9th. | — | Right thigh 95·6 | } | Thigh 0·8° warmer. |
| | | Left calf 94·8 | | |
| | | Right thigh 96·2 | } | Thigh 2·7 warmer. |
| | | Right calf 93·5 | | |
| ,, 10th. | — | Right thigh 93·6 | } | Calf 0·1 warmer. |
| | | Left calf 93·7 | | |
| ,, 15th. | — | Right thigh 95·5 | } | Equal. |
| | | Left calf 95·5 | | |
| ,, 18th. | — | Right thigh 96·2 | } | Thigh 0·7 warmer. |
| | | Left calf 95·5 | | |
| ,, 22nd. | — | Right thigh 93·5 | } | Thigh 2·1 warmer. |
| | | Left calf 91·4 | | |
| Ward 65·0. ,, 25th. | — | Right thigh 94·5 | } | Thigh 1·7 warmer. |
| | | Left calf 92·8 | | |
| ,, ,, 28th. | — | Left thigh 92·4 | } | Thigh 1·4 warmer. |
| | | Right calf 91·0 | | |
| ,, ,, 29th. | — | Right thigh 95·6 | } | Thigh 1·4 warmer. |
| | | Left calf 94·2 | | |
| ,, 61·0. Oct. 12th. | — | Left thigh 93·3 | } | Equal. |
| | | Right calf 93·3 | | |
| Small ward 70·0. Oct. 9th. | — | Left thigh 93·4 | } | Leg 2·2 warmer. |
| | | Right leg 95·6 | | |

The first six observations were made in warm weather, the windows of the ward were open, and the average temperature was about 70° Fahr. The observations were made in the morning between 8 and 10, excepting the last three, which were made in the afternoon. In the first ten observations the thermometers were placed in the same positions on thigh and calf. On thigh over the inner portion of the great extensor in its middle length; on calf over the inner portion of the gastrocnemius in its middle length. The thigh and calf of opposite sides were compared, lest the ligature over the thigh, though of slight tension, might affect the blood current to or from the leg. In the last observation the positions were changed respectively to the middle of the front of the thigh and to the middle of the front of the leg between the tibia and fibula. This observation was made in relation to statements of Dr. John Davy, and is excluded from the ten used for the establishment of an average.

It will be observed that in one instance the calf was warmer than the thigh to the extent of one tenth of a degree, that in two cases the temperature of the parts

compared were equal, and that in the remaining seven the thigh was from 0.7° to 2.7° Fahr. warmer than the calf. The average of the whole ten cases gave 1.07° in favour of the thigh, the average of numbers being 94.64° for the thigh, and 93.57° for the calf.

In a similar case, reported by me to the Society three years ago, the results were the reverse of what I have stated here, the calf being found in nine observations to be from 1.8° to 3.9° Fahr. warmer than the thigh.

The discrepancy of the two sets of observations has led me to take some care in exploring the conditions of surface thermometry, with the view of eliminating error and of satisfying myself as to the probable value of the tables in my first case.

I must admit in the first place that the first series of temperatures were not taken under a knowledge of many conditions which I have since found to be important, and that having been made sometimes on the exposed, sometimes on the partly covered, sometimes on the completely covered body, with only one instrument, and that a recording instrument, with an exposure of much less duration, they have much less of the claim of precision. But it must be remembered that under all these differences of method the results were actually more unvarying than under the uniform management of the second series.

It does not appear to me, indeed, that their value is lost if, after remembering their consensus, we recognise some important differences in the cases associated with the two series. The differences are :

1st. That while in the second case the duration of the disease appeared to have been twice as long as in the first the muscles had not grown to nearly an equal bulk.

2nd. That other muscles than those of the calf were affected with the hypertrophy in the second case and not in the first, indicating the presence of a much later stage of the disease.

3rd. That the information given by the microscope leads again to the inference that the muscles in the second

case have advanced considerably in a progress of degeneration which was not at all detected in the first.

The smaller bulk would probably indicate a less intensity or comprehension of the local mischief, and the degeneration would indicate that even if active hyperæmia had constituted an important element in the early stage the balance of nutrition had, later on, been reversed. It would follow that a smaller increase of temperature might in the beginning be looked for, and that in the later period now under observation a diminution proportionate to the amount of the degeneration might not improbably have occurred. In a word, the second series, though reversing the first, do not necessarily contradict them.

In this light it has seemed to me desirable to endeavour to learn what is the natural ratio of temperature between the calf as a mass and the thigh as a mass. The examination of this question shows many difficulties to exist. As an illustration of the difficulties I subjoin Dr. John Davy's table of temperatures obtained with the instrument already noted.* The observations were made on the naked body at 7 a.m., immediately on quitting bed. The temperature of the air of the room was 70°.

| | |
|--|------|
| At the central part of the sole of the foot | 90° |
| Between the malleolus internus and the insertion of the tendo Achillis, where the artery is felt | 93 |
| Over the middle of the tibia | 91·5 |
| Over the middle of the calf | 93 |
| Over the popliteal artery at the bend of the knee | 95 |
| Over the femoral artery in the middle of the thigh | 94 |
| Over the middle of the rectus muscle | 91 |
| Over the great vessels in the groin | 96·5 |
| Under the axilla, the whole surface of the bulb being exposed | 98 |

This, with some other determinations, occupied an hour, at the end of which time the sole of the foot gave no more than 85°, five degrees less than at first.

In these observations the middle of the calf gave a higher temperature than the middle of the rectus muscle,

* 'Account of Experiments on Animal Heat: Researches, Physiological and Anatomical,' 1839, p. 150.

and approached within one degree of the temperature of the skin over the femoral artery in the middle of the thigh. But if the observations were made in the order in which they are recorded it is clear that the conditions would not have been the same at or towards the end as at the beginning, since it is shown that the sole of the foot cooled no less than five degrees in the period of examination; and it cannot be assumed that the parts cooled altogether in the same ratio, it being quite possible that while the feet were cooler the thighs might have become warmer. For Mr. Garrod has given reason for stating it to be a rule that the application of warmth to the feet produces a lowering of temperature elsewhere; and I have myself made observations which confirm this and carry it further. I will quote one of these:

A well-nourished boy, of 16, having bared his legs, was covered with a blanket and a light shawl, and allowed to recline in a large easy chair for five minutes, the temperature of the room being 64.2° . He felt now comfortably warm. The thermometers applied for twenty-five minutes gave—

| | | | | | |
|--------------------|---|---|---|---|----------------|
| For the right calf | . | . | . | . | 93.5° |
| „ left „ | . | . | . | . | 91.7° |

although no difference of conditions, internal or external, was known to exist.

A hot-water bottle was then placed against the side of the left thigh, and while the thermometers were applied reversed to the calves, two others were placed over the inner part of the rectus in each thigh at the junction of the middle and lower thirds, the thermometer on the left thigh being thickly covered with cotton wool as well as being three and a half inches distant from the bottle. The fellow instrument was similarly covered. The readings at the end of thirty minutes were—

| | | | | | |
|--------------|---|---|---|---|----------------|
| Right thigh. | . | . | . | . | 95.1° |
| Left thigh . | . | . | . | . | 95.8 |
| Right calf . | . | . | . | . | 94.7 |
| Left calf . | . | . | . | . | 91.3 |

A sensation of warmth was experienced in the thighs, of cold in the calves, which also felt cool to the applied hand.

The application of warmth to the thigh was, therefore, followed by a fall of 0.4° in the calf of the same side, and a rise of 1.2° in the calf of the opposite side.

On the other hand, it is clear that no considerations of this kind will explain the difference in the temperature of the skin over the middle of the calf, 93.0° , as compared with the temperature of the skin over the tibia, 91.5° . The texture of the epidermis, thickness of subjacent fat, and nearness to large vessels will be among the circumstances thus causing such differences. These influences have been balanced by selecting for comparison in different limb-shafts parts agreeing as much as possible in these points; and when healthy people have been examined an average of 1.5° excess has been observed in the thigh as compared with the calf. The average of seven carefully selected cases among healthy people gave 93.87° for the thigh-muscles, and 92.30° for the calf-muscles. But it will be necessary to make many more comparative observations before their average can be regarded as correct, the variations being considerable, though with only one exception the excess was always on the one side. It will be seen that the average temperature differences in the case of W. Marshall were 1.07° or less than the difference in healthy persons.

Used with the precautions mentioned the thermometers I have described answer, I fully believe, the purpose in view, namely, the determination of the heat of the mass of flesh beneath the surface over which the pad is placed. But there are other cases in which the temperature of the surface is alone sought, or sought in comparison with the temperature of the depths. For such kind of investigation the use of some kind of thermopile and galvanometer would seem suitable; and, indeed, for very delicate observations of a comparative nature thermo-electrical apparatus suggests itself as most appropriate.

Thermo-electricity has, in fact, been already so applied, by Becquerel and Breschet, Helmholtz, Lombard, Gavarret, Montgomery, and others.

The original apparatus of Becquerel was a needle of copper soldered end to end with a needle of steel, so that their axes were continuous. This was either applied to a surface or thrust beneath, so that in either case the junction of the two metals was placed at the point to be examined as to its temperature. The free ends of the needles were connected with the poles of a very delicate galvanometer, the needle of which was left not quite perfectly astatic, and the actual temperature was estimated by comparing the deflection in each experiment with the deflection produced by immersion of a similar coupling in water of known temperature. The results were very interesting, but cannot be accepted as thoroughly accurate.

In using this kind of apparatus the experimenter is on the horns of a dilemma. He may use either a very simple thermopile of one pair of elements, and as a necessary associate a galvanometer of great delicacy, or he may use a thermopile of greater complexity and sensitiveness, in association with a less sensitive multiplier.

Error has to be guarded against in these appliances, both from the side of the pile and from the side of the galvanometer. In proportion as the galvanometer is made more sensitive it will tend to be affected by the currents produced by weak chemical actions, and by the currents existing in animal bodies. On the other hand, it is necessary to protect the thermopile from unequal heating, and, where comparative observations are made, to ensure perfect calibration of the two instruments, to maintain equal pressure, and ensure perfect equality of application.

I will take Dr. Montgomery's experiments recorded in p. 17 et seq. of the first volume of Holmes' 'System of Surgery.' They were comparative observations made with the view of determining whether heat were generated in inflamed parts. Small dart-like thermopiles, like those which I now exhibit, were used in conjunction with a

sensitive galvanometer. The darts were made of small bars of platinum and steel joined at an acute angle, and brought to a point at their junction. They were connected with each other, and then included in the galvanometric circuit. Being applied to the surface at different spots, or thrust, one into a vein, the other into an artery, they caused deflection of the galvanometer in one direction or another according as one was made warmer than the other.

Now, in thrusting a needle into the skin friction is involved, and if the skin at two points be of unequal toughness, if one needle be a little blunter or less polished, an element of disorder is introduced. Becquerel and Breschet take note of this as well as the next difficulty, that of currents being possibly produced by electrolytic action, a danger which is likely to be greater when bloods of different composition are investigated with the aid of very sensitive galvanometers. They suggest, accordingly, the varnishing of the needles (a precaution, indeed, which Dutrochet afterwards systematically used), but recognise the frictional error that may thus be introduced. Again, when a needle is introduced into the body it is apt by mechanical irritation, and, when composed of two metals, by galvanic stimulation, to cause contractions of muscles and currents in nerves. In preparing frogs for electrical investigation it will always be noticed that if a silver probe and the end of a pair of steel forceps come together under the lumbar nerves a sharp contraction of the limbs accompanies each contact.

I cite these points as illustrative of the difficulties which occur to me as having to be met in such investigations, and as grounds for my hesitation in making use of the same kind of apparatus for my own purpose.

If the surface alone is to be examined the proper compensation of the thermopiles is necessary, and is a matter involving great care and special arrangements. For it is unnecessary to remind the Society that in applying warmth to a thermopile, directly the rise of temperature travels beyond the midpoint between the poles reverse currents

are set up. It was probably to avoid this that Dr. Montgomery had the two piles which I now exhibit made so long; and it must be observed that this would prevent much error in short observations, but would not supersede the necessity of proper compensation.

My friend Dr. Kilner informs me that he has very nearly completed an electrical arrangement which will be available for clinical purposes. In this the distal end of the thermopile is immersed in a chamber containing water of known temperature, and in the same chamber is inserted at only a short distance the end of another pile, which is introduced into the circuit. As one pile becomes warm it raises the temperature of the water, and this in turn warms the second pile. The two currents then neutralise each other, and, as Dr. Kilner hopes to show, eliminate error. In such an instrument, combined with a suitable galvanometer, the indications of the needle can be measured and translated readily into the language of ordinary thermometry.

Supposing that such an instrument prove successful, and that the registration of surface temperatures becomes possible, and that observations may be made readily in clinical work, the difficulties on the physiological side will have to be borne in mind. The signs given by this or any other instrument must be subjected to criticism by the light of our knowledge of the influences effective in modifying local and comparative temperature in the body.

In conclusion, I have to acknowledge my great obligations to Dr. Charles, Dr. Twining, Mr. Nicholson, and Mr. Gulliver, who have assisted me in many ways, in relation to this case and the observations of temperatures generally.

ON A FORM
OF
CHRONIC INFLAMMATION OF BONES
(OSTEITIS DEFORMANS).

BY
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I HOPE it will be agreeable to the Society if I make known some of the results of a study of a rare disease of bones.

The patient on whom I was able to study it was a gentleman of good family, whose parents and grandparents lived to old age with apparently sound health, and among whose relatives no disease was known to have prevailed. Especially, gout and rheumatism, I was told, were not known among them ; but one of his sisters died with chronic cancer of the breast.

Till 1854, when he was forty-six years old, the patient had no sign of disease, either general or local. He was a tall, thin, well-formed man, father of healthy children, very active in both mind and body. He lived very temperately, could digest, as he said, anything, and slept always soundly.

At forty-six, from no assigned cause, unless it were that he lived in a rather cold and damp place in the North of England, he began to be subject to aching pains

in his thighs and legs. They were felt chiefly after active exercise, but were never severe ; yet the limbs became less agile or, as he called them, "less serviceable," and after about a year he noticed that his left shin was misshapen. His general health was, however, quite unaffected.

I first saw this gentleman in 1856, when these things had been observed for about two years. Except that he was very grey and looked rather old for his age, he might have been considered as in perfect health. He walked with full strength and power, but somewhat stiffly. His left tibia, especially in its lower half, was broad, and felt nodular and uneven, as if not only itself but its periosteum and the integuments over it were thickened. In a much less degree similar changes could be felt in the lower half of the left femur. This limb was occasionally but never severely painful, and there was no tenderness on pressure. Every function appeared well discharged, except that the urine showed rather frequent deposits of lithates. Regarding the case as one of chronic periostitis, I advised iodide of potassium and *Liquor Potassæ* ; but they did no good.

Three years later I saw the patient with Mr. Stanley. He was in the same good general health, but the left tibia had become larger, and had a well-marked anterior curve, as if lengthened while its ends were held in place by their attachments to the unchanged fibula. The left femur also was now distinctly enlarged, and felt tuberos at the junction of its upper and middle thirds, and was arched forwards and outwards, so that he could not bring the left knee into contact with the right. There was also some appearance of widening of the left side of the pelvis, the nates on this side being flattened and lowered, and the great trochanter projecting nearly half an inch further from the middle line. The left limb was about a quarter of an inch shorter than the right. The patient believed that the right side of his skull was enlarged, for his hats had become too tight ; but the change was not clearly visible.

Notwithstanding these progressive changes, the patient

suffered very little ; he had lived actively, walking, riding, and engaging in all the usual pursuits of a country gentleman, and, except that his limb was clumsy, he might have been indifferent to it. He had taken various medicines, but none had done any good, and iodine, in whatever form, had always done harm.

In the next seventeen years of his life I rarely saw him, but the story of his disease, of which I often heard, may be briefly told and with few dates, for its progress was nearly uniform and very slow. The left femur and tibia became larger, heavier, and somewhat more curved. Very slowly those of the right limb followed the same course, till they gained very nearly the same size and shape. The limbs thus became nearly symmetrical in their deformity, the curving of the left being only a little more outward than that of the right. At the same time, or later, the knees became gradually bent, and, as if by rigidity of their fibrous tissues, lost much of their natural range and movement.

The skull became gradually larger, so that nearly every year, for many years, his hat, and the helmet that he wore as a member of a Yeomanry Corps needed to be enlarged. In 1844 he wore a shako measuring twenty-two and a half inches inside ; in 1876 his hat measured twenty-seven and a quarter inches inside (Pl. I, fig 4). In its enlargement, however, the head retained its natural shape and, to the last, looked intellectual, though with some exaggeration.

The changes of shape and size in both the limbs and the head were arrested, or increased only imperceptibly, in the last three or four years of life.

The spine very slowly became curved and almost rigid. The whole of the cervical vertebræ and the upper dorsal formed a strong posterior, not angular, curve ; and an anterior curve, of similar shape, was formed by the lower dorsal and lumbar vertebræ. The length of the spine thus seemed lessened, and from a height of six feet one inch he sank to about five feet nine inches. At the same

time the chest became contracted, narrow, flattened laterally, deep from before backwards, and the movements of the ribs and of the spine were lessened. There was no complete rigidity, as if by union of bones, but all the movements were very restrained, as if by shortening and rigidity of the fibrous connections of the vertebrae and ribs.

The shape and habitual posture of the patient were thus made strange and peculiar. His head was advanced and lowered, so that the neck was very short, and the chin, when he held his head at ease, was more than an inch lower than the top of the sternum.

The short narrow chest suddenly widened into a much shorter and broad abdomen, and the pelvis was wide and low. The arms appeared unnaturally long, and, though the shoulders were very high, the hands hung low down by the thighs and in front of them. Altogether, the attitude in standing looked simian, strangely in contrast with the large head and handsome features.*

All the changes of shape and attitude are well shown in sketches from photographs taken six months before death (see Pl. I, figs. 1 to 3). Only the lowering of the necks of the femora is not shown. In measurement after death the axes of the shaft and neck of the right femur formed an angle of only 100° instead of 120° or 125° , and this change of shape added to the appearance of increased width of the pelvis.

But with all these changes in shape and mobility of the head, spine, and lower limbs, the upper limbs remained perfect, and there was no disturbance of the general health.

In 1870, when the disease had existed sixteen years, the left knee-joint was, for a time, actively inflamed and

* An attitude somewhat similar is given by a rare form of what I suppose to be general chronic rheumatic arthritis of the spine involving its articulations with the ribs. The spine droops and is stiff, the chest is narrow, the ribs scarcely move, the abdomen is low and broad, but there is no deformity of head or limbs.

its cavity was distended with fluid. But the inflammation soon subsided, only leaving the joint stiffer and more bent.

About this time some signs of insufficiency of the mitral valve were observed, but the patient now lived so quietly, and moved with so little speed, that this defect gave him no considerable distress.

In December, 1872, sight was partially destroyed by retinal hæmorrhage, first in one eye, then in the other,* and at nearly the same time he began to be somewhat deaf. In the summer of 1874 he had frequent cramps in the legs, and neuralgic pains, which were described as "jumping over all the upper part of the body except the head," but change of air seemed to cure them.

In January, 1876, he began to complain of pain in his left forearm and elbow which, at first, was thought to be neuralgic. But it grew worse, and swelling appeared about the upper third of the radius and increased rapidly, so that, when I saw him in the middle of February, it seemed certain that a firm medullary or osteoid cancerous growth was forming round the radius.

Still the general health was good. Auscultation could detect mitral disease, but the appetite and digestion were unimpaired, the urine was healthy, the mind as clear, patient, and calm as ever. As letters about him at this time said "his general health has been excellent;" "he is free from pain except in the left arm; he sleeps well, enjoys himself, and does not know what a headache is."

After this time, however, together with rapid increase of the growth upon the radius, there were gradual failure of strength and emaciation, and on the 24th of March, after two days of distress with pleural effusion on the right side, he died.

* Mr. Brudenell Carter saw him in January, 1873, and observed "the right retina sprinkled with small dots of arterial hæmorrhage, chiefly in parts remote from the centre;" and "there was no other change." The left retina was at this time healthy, but in February Dr. Clifford Allbutt found "several little plugs" in its vessels.

The body was examined five days after death, and showed no marked signs of decomposition. As it lay on a flat board its posture was remarkable, for the head was upraised to the level of the sternum, being supported by the rigid and arched spine, and the lower limbs, with the knees bent and stiff, rested on the heels and nates.

The pericranium, dura mater and all the substance of the brain appeared healthy.

The right pleural cavity contained at least a pint of pale serous fluid, with flakes and strings of inflammatory exudation. The lung was compressed, and in its pleural covering were numerous small nodular masses of pale cancerous substance. The proper pulmonary structure appeared healthy, and so did the left lung and its pleura, except that in the pleura and anterior mediastinum there were many small masses of cancer.

The heart was enlarged but thin-walled. The tricuspid and pulmonary valves and artery were healthy; the mitral valve was opaque, contracted, stiffened with atheromatous and calcareous deposits.

The aortic valves were slightly opaque but pliant, and both in them and in the first part of the aorta were numerous small patches of atheroma.

The liver and digestive canal and kidneys, examined externally, appeared healthy.

The right femur, the left tibia, the patellæ, and the upper part of the skull, were taken for separate examination, and will be separately described.

In the other bones of the skeleton, except the left radius, no signs of disease appeared externally, but I regret that they were not all more carefully examined, for I think that, at least in the clavicles and pelvis, some changes like those in the long bones of the lower limbs would have been found.

The upper third of the left radius was involved in a large ovoid mass of pale grey and white soft cancerous substance, similar to that of the nodules in the pleuræ and mediastinum, but with growths of bone extending

into it. The rest of the radius and the ulna appeared quite healthy.

Some nodules of similar cancerous substance were imbedded in the bones of the vault of the skull.

Microscopic sketches of these structures by Mr. Butlin are appended (Plate II, figs. 1-3).

The curvatures of the spine and its rigidity appeared due to shortening and hardening of its fibrous structures. The vertebræ appeared healthy; there was no appearance of overgrowth or anchylosis among them.

In no part, whether near or far from the diseased bones, was there an indication of any change of structure in skin, muscle, tendon, or fascia; but in the right hip-joint and in the left knee-joint there was some thinning and wasting of articular cartilage, such as one sees in chronic rheumatic arthritis. The other hip- and knee-joints and both ankle-joints were healthy.

In the arteries of the lower limbs there was extensive atheromatous and calcareous degeneration.

The enlargement of the skull may be estimated by comparison of the following measurements:

| | Diseased skull. | Average skull. |
|--|-----------------|----------------|
| Circumference at the level of the middle of the temporal fossa | 26½ in. | 21 in. |
| From occipital spine to base of nasal bones | 15 in. | 13¼ in. |
| From mastoid to mastoid process | 18½ in. | 15¼ in. |

All the sutures, at least all those of the vertex, were obliterated. The outer surface of the upper part of the skull was lowly bossed by the predominant thickening of the hinder part of the parietal bones. The thickness was in every part increased to the extent shown in these following measurements.

| | |
|--|--------------|
| In a median vertical section the thickness of the frontal bone was | 11—13 lines. |
| „ „ parietal | 14—16 „ |
| „ „ occipital | 8—12 „ |
| In a horizontal section, through the middle of the temporal fossa, the thickness of the frontal bone was | 8—9 „ |
| „ temporal „ | 6—9 „ |
| „ at their junction . | 2 „ |
| „ of the occipital bone was | 10—12 „ |

Comparing these measurements with those of average healthy skulls, it may be said that the bones of the vault of this skull were in every part increased to about four times the normal thickness.

The whole outer surface of the skull-cap was finely porous; in the least changed parts, such as the squamous bone, perforated with innumerable apertures for blood-vessels; in the most changed, finely reticulate, as with delicate cancellous and medullary texture.

The inner surface was comparatively smooth and appeared little changed, except by the enlargement of all channels and apertures for blood-vessels, and especially by the deepening of all the grooves for the middle meningeal artery and its branches.

On the cut surface, in the median vertical section, that which might be regarded as the altered internal table of the skull was a layer, having a very unequal thickness varying from two to six lines, consisting of hard white bone, close-textured, in some parts porous or finely reticulate, in more looking compact and dense like limestone or white brick (Pl. V).

The rest of the thickness of this part of the skull, representing probably the altered diploë and outer table, was made up of bone in various degrees porous, cancellous, or cavernous, with spaces filled with soft reddish substance, a kind of medulla. Its surface was covered with a very thin layer, a mere coating of more finely porous bone.

In the horizontal section, at the level of the upper part of the squamous bone, the same altered characters were observable, but a larger proportion of the substance of the skull was finely porous or reticulate.

By the cavities in the skull-cap in which cancerous growths were lodged the structure of the bone was neither more nor less altered than in other parts.

A portion of sphenoid bone showed changes of structure very similar to those already described, but with a much more uniform and regular finely porous condition.

The bones of the face were not uncovered, but they

showed, neither to sight nor touch, any appearance of disease; not a feature was unnatural.

The conditions of all the long bones were so similar that one description may serve for the altered structure of both femora and tibiæ.*

The periosteum was not visibly changed, not thicker or more than usually adherent.

The outer surface of the walls of the bones was irregularly and finely nodular, as with external deposits or outgrowths of bone, deeply grooved with channels for the larger periosteal blood-vessels, finely but visibly perforated in every part for transmission of the enlarged small vessels. Everything seemed to indicate a greatly increased quantity of blood in the vessels of the bone.†

The medullary structures appeared to the naked eye as little changed as the periosteum. The medullary spaces were filled with soft, yellow, ruddy, and bright crimson medulla, of apparently healthy consistence. The medullary laminae and cancelli had a normal aspect and arrangement, and in the shafts of the long bones the medullary spaces were not encroached upon.

The compact substance of the bones was, in every part, increased in thickness. Taking, for example, the femur, the thicknesses of its walls and those of a healthy femur of about the same length and age are compared in the following tables.

| | Healthy | Diseased. |
|---|-------------------------------|-------------|
| | Lines. | Lines. |
| Thickest parts of the wall . . . | 3—6 | 6—10 |
| Articular covering of head, about . . | $\frac{1}{4}$ | 3—10 |
| Wall of neck, about . . . | $\frac{1}{4}$ —3 | 4—6 |
| Wall of the trochanter major, about . | $\frac{1}{4}$ — $\frac{1}{2}$ | 3—5 |
| Articular covering of the condyles, about . | $\frac{1}{4}$ — $\frac{3}{4}$ | 3—5 |
| Lateral walls of the condyles . . . | $\frac{1}{6}$ — $\frac{1}{4}$ | 2 and more. |

Changes in similar proportions were found in the walls

* Their changes are shown in Pl. IV. The specimens are in the Museums of the Royal College of Surgeons and of St. Bartholomew's Hospital.

† But see p. 47 in the account of the microscopic examination.

of the tibia. In the patellæ the walls were from three to five lines thick.

The thickening of the walls of the shafts of the bones appeared due chiefly to outward expansion and some superficial outgrowth. In some places there were faint appearances of separation of parts of the outer layers of the walls, and of these becoming thick and porous, while the corresponding parts of the inner layers were less changed; but in the greater part of the walls the whole construction of the bone was altered into a hard, porous or finely reticulate substance, like very fine coral. In some places, especially in the walls of the femur, there were small, ill-defined patches of pale, dense, and hard bone looking as solid as brick.

In the compact covering of the articular ends of the long bones, and in those of the neck and great trochanter of the femur, and in the patellæ the increase of thickness was due to encroachment on the cancellous texture, as if by filling of its spaces with compact porous, new-formed bone.

Mr. Butlin was so good as to make careful microscopic examination of the diseased bones, and to give me the following report on them, together with the annexed drawings of their minute structure.

“Microscopical examination was made of sections cut from the skull and from the tibia, some of them from the recent bones, but the majority of them from portions of bone deprived of earthy salts and rendered sufficiently soft to be cut with a razor. The appearances observed were essentially the same in both bones, but most of the drawings and description were taken from the tibia, the sections of which were much clearer than those of the skull.

“The examination was conducted from a twofold point of view: first, to discover the changes which the bone had undergone; second, to discover, if possible, the nature of the process which had led to such changes.

“With a low power the number of Haversian systems

and canals in any given section was seen to be much diminished (Plate II, fig. 8; Plate III, fig. 9). The space between the Haversian canals was occupied by ordinary bone-substance, containing numerous lacunæ and canaliculi. The Haversian canals were enormously widened, many of them were confluent, and thus the appearance of a number of communicating medullary spaces was obtained, an appearance which was rendered still more striking by the presence in the canals of a large quantity of ill-developed tissue in addition to the blood-vessels (Plate II, figs. 4—6). With a high power the contents of the Haversian canals were seen to consist generally of a homogeneous or granular basis, containing cells of round or oval form about the size and having much the appearance of leucocytes. Larger nucleated cells were also present, and fibres or fibro-cells, sometimes in considerable quantity. Myeloid cells were occasionally observed, but they were not plentiful; fat also existed in many of the larger spaces, especially in the skull. The vessels were usually small compared with the channels in which they ran; indeed, they did not seem to be much larger than those of normal bone (Plate II, fig. 6). The walls of some of the canals were lined by a single layer of osteoblasts, a condition precisely similar to that observed in the normal ossification of bone in membrane. The presence of new bone was most evident in the periosteum of the tibia, external to the ordinary compact layer of the shaft (Plate II, fig. 7). This external layer was, of course, but thin, and was much softer and less developed than the cortex of the bone from which it sprung; it evidently was not nearly sufficient to account for the great increase in the diameter of the tibia. From the diminution in size of the medullary canal it was thought that a similar recent formation of bone would be found on its outskirts, but this expectation was not justified by observation.

“With a medium power the number of (Plate III, fig. 12) lamellæ surrounding the Haversian canals was

easily seen to be not larger than in normal bone, whilst the arrangement of the intervening space was most complex and totally different from that of healthy bone. The lacunæ and canaliculi throughout the sections did not strikingly differ from those of ordinary bone."

I am indebted to Dr. Russell for the following chemical analysis of portions of the diseased skull and tibia, and of a healthy tibia in comparison with them.

| | Skull. | Tibia. | Normal tibia. |
|---|--------|--------|---------------|
| Inorganic constituents (Ash) . . . | 60.59 | 61.22 | 63.62 |
| Organic " . . . | 39.41 | 38.78 | 36.38 |
| Phosphoric acid (P_2O_5) . . . | 22.76 | 25.45 | 25.50 |
| Carbonic " (CO_2) . . . | 3.59 | 3.95 | 3.59 |
| Fat . . . | 6.83 | 3.45 | — |
| Moisture in the sample (dried at $115^\circ C.$) . | 15.49 | 11.83 | 9.73 |
| The CO_2 calculated as calcium carbonate ($CaCO_3$) . . . | 8.17 | 8.99 | 8.16 |
| The P_2O_5 calculated as calcium phosphate ($Ca_3P_2O_8$) . . . | 49.70 | 55.56 | 55.66 |
| Specific gravity . . . | 1.895 | 1.889 | 1.886* |

Cases of the disease which I have described are so rare that I believe no one has seen a sufficient number of them to enable him to distinguish this disease, either clinically or anatomically, from some which seem like it. Specimens illustrating it are commonly included under a general name of hyperostosis, osteoporosis, senile rachitis, or the like. But I hope that, if I add to the description I have just given some notes of similar cases which I have seen or found on record, the disease may be so distinguished as to deserve in pathology a separate place and name.

CASE 2.—Some ten years ago I saw a gentleman, between fifty and sixty, very active, tall, thin, and muscular, a master of hounds. For many years before his death he had curvature of the thighs and legs, exactly like that

* Specific gravity of normal skull 1.990.

already described, and stooping of the spine. The changes of the limbs were attended with severe pains, which he used to relieve with hard rubbing, but the general health was unimpaired. In the last years of his life the upper part of his right humerus became very large, and as he was riding and suddenly raised his arm the bone broke near the shoulder. The evidence of large tumour now became clear, and I amputated the arm at the shoulder-joint. The tumour was well marked and very vascular medullary cancer investing and infiltrating the upper part of the humerus. The rest of the humerus was healthy, and the fracture, which was just below its neck, was evidently due to muscular force acting on its structures spoiled by the cancerous growth. He died a few days after the operation, but was not examined after death. The similarity of his case with that which I have described is, I think, certain.

CASE 3.—I saw, with the late Dr. Brinton, a gentleman between forty and fifty who may be still living. He was a sturdy and quite healthy man; his tibiae were curved and enlarged exactly like those in the first case and he had similar pains, but there was more thickening of periosteum and an appearance of more external formation of bone. He was treated with iodide of potassium and many other things as for periostitis, but without avail.

CASE 4.—A case is recorded by Dr. Wilks in the 'Transactions' of the Pathological Society,* and through the kindness of Sir William Gull, whom the patient occasionally consulted, I am enabled to add some facts to those in Dr. Wilks's report, and to show photographic portraits.

A summary of Dr. Wilks's report is that the patient was sixty when he died. Signs of the disease, beginning with pains like those of rheumatism in the legs, were first observed fourteen years before his death. It was soon found that the tibiae were enlarged, and in subsequent

* Vol. xx, p. 273, 1869.

years the cranium and nearly all the bones of the skeleton underwent similar changes. About a year before death the general health began to suffer from the thorax having become implicated in the disease. Gradually the chest became more contracted and at last quite fixed; the breathing became more difficult until at last the respiratory apparatus altogether stopped.

Sir William Gull's notes tell that the patient consulted him when fifty-six years old, and said that he first noticed enlargement in the left tibia when he was forty-five years old; that he had seven brothers well and strong, and was eldest in the family. He complained chiefly of weakness, inability to make exertion, feeling of nervousness with occasional vertigo, shortness of breath, stiffness in neck, hoarseness and feebleness of voice. His general health was good; he was not much troubled with pain anywhere; but had occasional strange sensations about the head, and much cough. His height, when a young man, was five feet three and a half inches, now four feet eleven and a half inches. The urine was normal and of normal colour. The cranium was enlarged and thickened; the clavicles much thickened, as also the long bones; the phalanges and facial bones and perhaps the lower jaw were not altered. The ribs were thick and immovable, as was also the sternum. There was general dulness over the chest on percussion. The respiration was chiefly diaphragmatic.

Less than a year before the patient's death Sir William Gull recorded that he was breathless, and had occasional attacks of mental confusion in which he remarked that he could not understand the sense of words. His voice was hoarse and feeble, and the hyoid bone seemed thickened. The head had continued to enlarge, and he maintained that he was still losing in height. The neck was fixed, and somewhat forward. All the viscera appeared normal. The urine, repeatedly examined, was always found normal, and of normal colour.

The record of the post-mortem examination by Dr. Goodhart leaves no doubt that the disease in this case

was the same as that which I have described, and it may be important that this patient also had cancerous disease. "A growth . . . corresponding to the growth described as epithelioma of the arachnoid surface of dura mater," grew from the inner surface of the dura mater, was as large as a chestnut and made a pit in the brain near the left Sylvian fissure.

The description of the changed structure of the bones, for which I may refer to the 'Pathological Transactions,' seems to me to indicate that the disease was more advanced in the direction of degeneracy than that which I have described, or that it had not been in any degree repaired.

CASE 5.—I owe to Mr. Bryant the opportunity of seeing a similar case which was under his care in Guy's Hospital, and of which Mr. Viney was so good as to give me notes.

The patient was a carpenter, sixty years old, a hard-working married man, and had seven children. When about sixteen years old he had a slight attack of gonorrhœa, but without sores, and no history of syphilis could be learned. When thirty-five years old he received an injury to his pelvis. Shortly after this he had trouble with his bladder, which became much distended; a large quantity of clotted blood was washed out. He lay in bed for this six weeks, and at the end of three months was able to go to work again.

For the last five years he had been troubled with gout in his left great toe. His father suffered from this. The attacks had been short; a few days' rest always sufficed for recovery.

About three years before admission he first felt pains of a shooting description about the tendons of the popliteal space, whenever he straightened his legs. At this time also he first noticed a swelling of the legs, which began at the ankles. These symptoms, without his taking any special notice of them, continued for about a year.

In the last year and a half the tibiæ had become much swollen and curved forwards, and on account of the pain

he had in them from standing he had been obliged to give up his regular work. Until admission he did not notice anything wrong with his other bones, but he had lost about half an inch in height.

The tibiæ presented a marked curve forwards. The anterior border of each was rounded to a very marked degree, so that it could not be felt at all distinctly. The right tibia was slightly larger than the left. The inner surface of each measured about four inches at its widest part. The veins above the ankle were in a varicose condition.

The fibulæ were very much enlarged; the femora enlarged in their shafts and bowed outwards. The great trochanter was drawn up to the level of a vertical line drawn from the anterior superior spinous process of the ilium to the horizontal line of the body, instead of being about two and a half inches below this line. The patellæ were little larger than natural.

The bones of the upper extremity were enlarged, but not to so marked a degree as those of the lower. The enlargement was most marked in the humeri and the left was thicker than the right. He could not straighten his arms, probably owing to the enlargement of the olecranon. In the clavicles the natural curves were very much increased and the bones thickened, the left more so than the right. In the scapulæ the spines and acromion processes were very much enlarged.

The chest was slightly flattened from side to side, but moved fairly whilst breathing. The ribs on the right side were slightly larger than those on the left.

There was a general curve backwards from the cervical to the dorsal vertebræ, so that the patient's usual position in bed was with his head bent forwards, and his legs in a semi-extended position.

The bones of the hands and feet did not seem to have shared in the general thickening.

There seemed to be a slight thickening about the external protuberance of the occipital bone, but there was no other evidence of the cranial bones being involved.

The patient had cold perspirations over his legs in the evening. His urine had a specific gravity of 1014, was strongly acid, contained a little albumen but no excess of phosphates.

[Six months later Mr. Bryant told me that this patient's bones were still enlarging, and that there were evidences of enlargement of the skull.]

I have looked for records of cases similar to these in nearly every work that seemed likely to contain them, but in vain. I have found only three cases, and the first two of these are doubtful.

Saucerotte* relates the case of a man who died at forty and in whom all the bones, those of the head, face, orbits, ribs, vertebræ and limbs had begun to enlarge about seven years before death. He increased in weight from 119 livres to 168, wholly from increase of bones; he had rheumatic pains; for a time sleepiness, oppression at the chest, and very small pulse; but these passed-by and he died with some acute illness. No examination was made.

Rullier† tells of a man, aged seventy-eight, who died in the Hôtel Dieu of empyema. He had previously been in good health, and nothing had indicated any derangement of cerebral function. The skull was very large, osteoporotic, and heavy, and, except the lower jaw, all the bones of the face were healthy. The ribs were thicker and larger than usual; the sternum narrow and very thick; the pelvic bones changed like those of the skull. The clavicles were thick, curved, and solid. The other bones were healthy.

Wrany‡ has fully described the condition of the bones in a case of spongy hyperostosis of the skull, pelvis, and left femur, taken from a woman fifty years old, of whom, however, nothing is told but that she died of pyæmia, and

* 'Mélanges de Chirurgie,' Paris, 1801.

† 'Bulletin de l'École de Médecine de Paris,' t. ii, p. 94, 1812.

‡ 'Prager Vierteljahrschrift,' 1867, B. i, p. 79.

that she had "spongy hyperostosis of the skull with atrophy of the facial skeleton, spongy hyperostosis of the vertebral column, pelvis, and left femur, with elongation of the latter bone; kyphoscoliosis of the upper dorsal part of the spine; pelvic abscess; emphysema and œdema of both lungs, abscess of the left; marasmus."

I cannot doubt that this disease was the same as I have here described, and the paper is valuable, both for the many signs indicated in it that the bones softened and yielded to pressure in the early part of the disease, and for the careful comparison of the distortion of the pelvis with the dissimilar distortions in rickets and mollities ossium. The spine was very curved; the chest small and too arched; the whole trunk very short.

From these cases which, though few, are well marked and in some chief points uniform, as well as from a recollection of two more of which I have no notes, I think we may believe that we have to do with a disease of bones of which the following are the most frequent characters:—It begins in middle age or later, is very slow in progress, may continue for many years without influence on the general health, and may give no other trouble than those which are due to the changes of shape, size, and direction of the diseased bones. Even when the skull is hugely thickened, and all its bones exceedingly altered in structure, the mind remains unaffected.

The disease affects most frequently the long bones of the lower extremities and the skull, and is usually symmetrical. The bones enlarge and soften, and those bearing weight yield and become unnaturally curved and misshapen. The spine, whether by yielding to the weight of the overgrown skull, or by change in its own structures, may sink and seem to shorten with greatly increased dorsal and lumbar curves; the pelvis may become wide; the necks of the femora may become nearly horizontal, but the limbs, however misshapen, remain strong and fit to support the trunk.

In its earlier periods, and sometimes through all its

course, the disease is attended with pains in the affected bones, pains widely various in severity and variously described as rheumatic, gouty, or neuralgic, not especially nocturnal or periodical. It is not attended with fever. No characteristic conditions of urine or fæces have been found in it. It is not associated with syphilis* or any other known constitutional disease, unless it be cancer.

In three out of the five well-marked cases that I have seen or read-of cancer appeared late in life ; a remarkable proportion, possibly not more than might have occurred in accidental coincidences, yet suggesting careful inquiry.†

The bones examined after death show the consequences of an inflammation affecting, in the skull the whole thickness, in the long bones chiefly the compact structure, of their walls, and not only the walls of their shafts but, in a very characteristic manner, those of their articular surfaces.

The changes of structure produced in the earliest periods of the disease have not yet been observed, but it may certainly be believed that they are inflammatory, for the softening is associated with enlargement and with excessive production of imperfectly developed structures, and with increased blood-supply. Whether inflammation in any degree continues to the last, or whether, after many years of progress, any reparative changes ensue, after the manner of a so-called consecutive hardening, is uncertain.

The inflammatory nature of the disease is evident also in the changes of minute structure in the affected bones.‡

* There has not only been no history of syphilis in any of the cases, but no known syphilitic changes have been observed in any patient.

† See, also, Sandifort, quoted at p. 61 ; Museum of St. Bartholomew's, ser. i, 111 and 112, sections of a femur, large, curved, porous, with a tumour growing around its shaft ; and 49, a hyperostotic skull from a man who died with cancerous disease of the eyeball, heart, and other organs ; and Museum of Guy's Hospital, specimens of symmetrical osteoid cancer of the ilia, with cancer of the spine and cranium, associated with hypertrophy of the cranium. Dr. Goodhart was so good as to give me a report of this case.

‡ And this is also the opinion of Wrány, l. c.

On these Mr. Butlin writes, "With regard to the nature of the process by which these changes were accomplished, there are probably only three things which could produce so great an increase in the size of a bone, namely, new growth (tumour), hypertrophy, and chronic inflammation.

"The first of these may be at once set aside as out of the question.

"Nor is the second much more probable than the first, for the process is evidently no mere hypertrophy. The whole microscopical architecture of the bone has been altered; the structure appears to have been almost entirely removed and laid down afresh on a different plan and in a larger mould.

"Of the three causes chronic inflammation alone remains, and upon examination one or two facts will be found to bear strongly upon the theory of this being essentially an inflammatory disease. Not only the absorption of the old structure which has taken place, but also the manner of this absorption, point to its inflammatory nature. Traces of this are not, of course, always discernible, as the process is almost everywhere far advanced. But still careful observation not uncommonly discovers that the sides of the widened canals, instead of being smooth and even (Plate III, fig. 10), are eaten out in a series of curves or concavities with the production of what are called Howship's lacunæ, so characteristic of inflammation. The tissue contained in the canals, too, almost precisely resembles the tissue found in the spaces of inflamed bones, only differing from it in being generally more fibrillar and less rich in cells, a fact easily to be accounted for by the very long duration of the disease and the general tendency towards organisation which was displayed throughout. The apparent cessation of the process of absorption and the gradual process of repair may be regarded as still further leading towards the same conclusion.

"Further than this the microscopical observations do not extend."

The chemical analysis by Dr. Russell may be regarded as confirming this conclusion. It shows, at least, that there is no such change of composition in the bone as would be expected in any merely degenerative softening.

Holding, then, the disease to be an inflammation of bones, I would suggest that, for brief reference, and for the present, it may be called, after its most striking character, *Osteitis deformans*. A better name may be given when more is known of it.

It remains that I should point out the distinctions between this disease and the several forms of hyperostosis, osteoporosis, and other diseases among which it has been confused.*

1. Among cases of hyperostosis are included those of simple overgrowth or hypertrophy of bones in adaptation to increase or change of office. The distinction of these from any form of disease is plain enough; they show a mere increase of natural structure.†

2. Scarcely different from these and as easily distinguished are the hyperostoses, best seen in the skull, in which the bones have more than normal thickness, hardness, and weight, and marks of greater vascularity, yet preserve a just relation of their several parts and a scarcely changed structure. They probably illustrate the effects of simple inflammation of bone recovered from.‡

3. A group of hyperostoses consists of those cases in which bones are enlarged in consequence of an increased supply of blood or lymph. Such a case is that recorded by Dr. Day§ in which the bones of a boy's limb with obstructed lymphatics are much longer than those of the sound limb;|| and such are all those in which bones near

* Many of the statements here made are derived from the examinations of the collections of diseased bones in the College of Surgeons and St. Bartholomew's Hospital, which I made while writing the catalogues of their pathological museums.

† Mus. Coll. Surg., 379, 380, 2838, 2839, 2842, 2843, &c.

‡ Mus. Coll. Surg., 2840, 2841.

§ 'Transactions of the Clinical Society,' vol. ii, p. 104, 1869.

|| Broca, 'Des Anévrysmes,' 8vo, p. 76, 1856, gives a case of femoral arterio-venous aneurism attended with considerable elongation of the limb.

inflamed joints, or with partial necrosis, or in limbs long hyperæmic, from whatever cause, grow in length and circumference till they considerably surpass the bones of the healthy limb.* These are easily distinguished. They have not signs of disease proper to themselves; they occur in the young alone; they may present a healthy texture, or one only slightly changed as by partaking of the adjacent inflammatory process; and with the exception of the tibia they do not become deformed. The tibia, when it lengthens more than the fibula, is almost compelled to curvature by the fixed unyielding attachment of its ends;† and the curve is usually similar in shape and direction to the curve of the tibia in the osteitis deformans. But there is no other likeness between the two conditions.

4. A very large number of cases of hyperostosis are consequences of inflammations of bone; some of simple inflammation, others of scrofulous, syphilitic, or gouty inflammation. It is not necessary here to distinguish these from each other,‡ but there are sufficient signs for the distinction of all from the osteitis deformans.

It is clear that the summary which I have given of the clinical characters of this osteitis would not tally with that of any case of simple osteitis, such as might ensue in a healthy person after injury, or in the neighbourhood of a sequestrum; and the clinical difference is as complete between it and any case that could justly be regarded as strumous or syphilitic or gouty osteitis.

The anatomical differences are as well marked: chiefly

* I believe these were first described by Mr. Stanley, 'On Diseases of Bones,' p. 20, *et seq.*, and myself, 'Lectures on Surgical Pathology,' p. 64, ed. 3, and in the catalogues already referred to. Langenbeck has published a very interesting paper on them in the 'Berliner Klin. Wochenschrift,' 1869, No. 26. Cases are also cited from Weinlechner, Schott, and Bergmann, in Virchow and Hirsch's 'Jahresbericht für 1869.'

† Such curved tibiæ are in the museum of St. Bartholomew's, Nos. A. 3, A. 46.

‡ An attempt to do so is made in the pathological catalogue of the College of Surgeons.

in the facts that in these inflammations the bones do not become curved* (unless in the case of the tibia already explained); that they commonly display much more considerable external periosteal outgrowths or deposits, as if from a greater participation of the periosteum in the inflammatory process; that the rarefied or, it may be, porous structure of the swollen shafts of bones usually shows appearances of separation and expansion of the component layers; that the medullary canals are commonly invaded by the thickening walls, or are as much changed as the walls themselves; that the whole length of a bone-shaft is very rarely affected; and that the thin articular layers of bones are, I believe, never thickened as they are in the osteitis deformans.†

It may be added that it is very improbable that any form or degree of scrofula or syphilis or gout should exist in bones or any other textures for ten or more years without affecting other parts and without impairing the general health. The retention of good general health during many years of localised disease is, indeed, one of the most striking characters of the osteitis deformans. The only parallel known to me is in the rheumatoid or chronic rheumatic arthritis, and the likeness between the two in this respect may suggest that they are nearly related; yet they are not found concurrent. In the case that I have related the amount of chronic rheumatic arthritis was trivial, and (which is more important) in all the records and specimens of the arthritis which I have seen I have not found an instance in which there were any of the morbid changes characteristic of the osteitis.‡

5. There are, I think, only two other diseases, namely,

* The absence of curving in bones around sequestra is remarkable, for they are long and often acutely inflamed, and those of the lower limbs are commonly used and bear weight.

† Among the specimens in which these changes may be studied are, in the College Museum, Nos. 3085, 3089, 598, 3090, 3091; in the museum at St. Bartholomew's, A. 1, and ser. i, 56, 132, 138, 196—198.

‡ There is not even any mention of them in Mr. R. Adams's elaborate 'Treatise on Rheumatic Gout,' 1873, 8vo and folio.

rachitis and osteomalacia, from which it can be necessary to discriminate the osteitis deformans, and the differences between them are very wide. They have scarcely a feature in common except that in all of them the bones bearing weight become curved or misshapen, and the spine is usually deformed, and the skull may become very thick and porous. But in rachitis the bones are too short, not too long; too small, not too large; and their curvatures are quite unlike those of the osteitis. And in the osteomalacia the walls of the bones become exceedingly thin, wasting with an acute atrophy; and when they yield it is not with regular curving but with angular bending or breaking. By these and many other differences, as well clinical as anatomical, the diagnosis of the osteitis from rachitis and osteomalacia is sufficiently clear. With rachitis it may be judged to have no affinity whatever; with osteomalacia only so much as may exist between a chronic inflammation and an acute atrophy of any part. Yet by one character which all these three diseases have or may have in common, namely, the osteoporosis of the skull, they are constantly confounded in museums, if not in practice, with each other, and with diseases different from them all.

The study of the osteitis deformans led me to learn what I could of the various recorded descriptions of large, thick, and porous skulls often found in museums. Nearly every large museum contains one or more specimens of such skulls whole or in fragments. They are all big, thick, porous, or spongy, with obliterated sutures and wide apertures and grooves for blood-vessels. Very few of these specimens have any life-histories; they are all, in many respects, alike and usually are all named alike. Many of them it may be impossible to name or classify without much better knowledge of them than may now be had, but I believe that among them are the results of several different diseases; and it may save some trouble to future students if I refer to some of the specimens and records which have led me to this belief.

1. Some are examples of the osteitis deformans which I have described.*

2. Some are derived from cases of osteomalacia. Mr. Durham† has written on these, and Mr. Solly's‡ well-known paper gives a good instance of them. In general, I think that these may be distinguished, at least in the recent state, by their softness and lightness; the abundance of soft medulla contained in them, and the comparative brittleness of the bones when dry.

3. Some are from rachitis; they are, unless after recovery and repair, very light, almost friable, and on their surface not porous, but like fine cloth or felt.§ Like these are the skulls of some lions and monkeys which have died young, in confinement, of what is considered rickets. A collection of these skulls and other similarly diseased bones in the college museum|| deserves careful study, especially because of their likeness to the cases included in the next group.¶

4. These are the results of a disease of early life, sometimes even of childhood, in which all the bones of the face as well as those of the cranium are affected, and, it is said, the bones of the limbs. All the affected bones, facial as well as cranial (and herein is a clear ground of diagnosis), become hugely thickened, porous, or reticulate.

* To those already referred to these, I think, may be added: Sandifort, 'Museum Anat. Acad.,' Lugd.-Bat., fol., 1835, vol. i, p. 142, vol. ii, tab. xiii, Skull of a man forty-three years old, with a "fungus" over the left orbit (? a cancerous growth). Other similar skulls are here referred to. Similar specimens are, probably, Nos. 2840 and 2858a in the College Museum; and, more uncertainly, 2841 and 2858, which, perhaps, belong rather to the fifth group.

† 'Guy's Hospital Reports,' ser. iii, vol. x, 1864.

‡ 'Med. Chir. Trans.,' vol. xxvii, p. 435, Mus. Coll. Surg., 395.

§ See Mus. Coll. Surg., 390—394, 2844, and 2857. I believe that Huschke, 'Ueber Craniosclerosis,' 1858, quoted by Virchow, contains facts on the rachitic osteoporosis, but I have not been able to refer to it.

|| Nos. 386—388, 2854—2856, 2855A, &c.

¶ Although bones such as these are not described by Paul Gervais, yet his paper quoted below should be studied on all that relates to hyperostosis in animals.

The whole skull is very large, clumsy, and featureless. Commonly the cranial cavity is diminished. The orbital and nasal cavities are contracted, the antra are often filled, by the ingrowth of their several walls; the apertures for nerves are narrowed or obliterated.*

Of these cases, which are among those named by Virchow† Leontiasis ossea, the best are related by Ilg‡ and Jadelot.§ Their descriptions are very scanty, yet they give sufficient facts to distinguish the disease by their account of the cerebral symptoms associated with it. In Ilg's case, for example, the patient who died at twenty-seven, after seventeen years' disease, had amaurosis, epilepsy, severe general headache, delirium, convulsive attacks, and at last total deafness, witlessness, difficulty of swallowing, and loss of smell.

5. Some cases, perhaps not different from these, though they have occurred in later life, are those by Schützenberger,|| Otto,¶ and Wraný.**

6. And, lastly, there are cases not so much of thickening

* Among the casts in the museum at St. Bartholomew's, No. 10, is that of a skull affected with this disease, and in ser. i, 36 are fragments of a bone, which, I think, may be referred to it.

† 'Die krankhaften Geschwülste,' B. 11, 1864-5. I need not say that this contains a very complete account of all forms of overgrowth of bone.

‡ 'Einige Anatomische Beobachtungen,' 4to, Prag, 1821.

§ Quoted by Ilg from Meckel. The best of many accounts of this specimen is given by Paul Gervais, "De l'hyperostose chez l'homme et chez les animaux," in the 'Journal de Zoologie,' t. iv, 1875. He has carefully re-examined the skull and face and described them.

|| 'Gazette Médicale de Strasbourg,' and in Canstatt's 'Jahresbericht für 1856,' B. iii, 34, with references to cases by Breschet and Nélaton.

¶ Otto, 'Neue seltene Beobachtungen,' 4to, 1824, p. 2. Both head and face are affected; the bones are described as, after softening, very hard, dense, and almost ivory-like. Six hyperostotic skulls are mentioned in his 'Neues Verzeichniss der Anat. Sammlung zu Breslau,' 1841.

** Wraný, "Hyperostosis maxillarum," in 'Prager Vierteljahrschrift,' 1867, B. 1, similar affections of the facial and cranial bones, with cerebral symptoms. Doubtful cases by Ribelt are quoted by Ilg, l. c.; Malpighi, 'Opera Posthuma,' 4to, Amstel., 1700, p. 68; Kilian, 'Anat. Unters. über den neunten Hirnnervenpaar,' Pesth, 4to, 1822, p. 133; Quekett, reported by Hewett, 'Medical Times and Gazette,' Sept. 8th, 1855, p. 229.

of the cranial and facial bones as of enormous bossed and nodular hard bony outgrowths overspreading them or projecting from them. The leading case among these is that published in the 'Transactions' of the Pathological Society by Dr. Murchison,* with a report on the specimens by Mr. De Morgan and Mr. Hulke.† The disease in which the facial more than the cranial bones are affected is clearly distinct from any of the foregoing, or if it be in any way connected with them, especially with those of the fifth group, may be regarded as transitional from them to the exostoses, especially the massive tuberous and bossed ivory exostoses, which grow on or among the bones of the face and skull. The same approach to the character of hard exostoses is shown in the disease of the fibula in Dr. Murchison's case, a section of which, from the museum of the Middlesex Hospital, is now before the Society.

* Vol. xvii, 1866, p. 243.

† Similar cases are illustrated by Forcade, quoted in Virchow's 'Die krankhaften Geschwülste,' B. 2, p. 22; Weber, from a specimen in the Dupuytren Museum, in v. Pitha and Billroth's 'Handbuch,' B. 3, Abth. 1, Lief ii, p. 257; Howship, 'Practical Observations in Surgery and Morbid Anatomy,' 1816, p. 26; Adams in 'Trans. of the Pathological Society,' vol. xxii, p. 204, 1871; Lysthay, in Canstatt's 'Jahresbericht für 1858,' Mus. Coll. Surg. Eng., 3093. Virchow has a full account of nearly all these cases, and of the analogies of the disease with elephantiasis of soft parts.

DESCRIPTION OF PLATES I TO V.

Chronic inflammation of bones (Osteitis deformans).

PLATE I. See pp. 39-40.

Figs. 1—3. From photographs of the patient (Case 1) taken six months before death.

Fig. 4. From photographs of the same patient's cap worn in 1844, and hat worn in 1876.

PLATE II. See pp. 42-3 and 46.

Figs. 1, 2. From tumour of forearm. Fig. 1. Oc. 3, obj. 4. \times about 62. Fig. 2. Oc. 3, obj. 7. Tube drawn out. \times 260.

Fig. 3. From secondary tumour of pleura. Oc. 3, obj. 7, t. dr. o. \times 260. See p. 42-3.

Figs. 4, 5. To show tissue in widened canals of tibia (4) and skull (5). Oc. 3, obj. 7, t. d. o. \times about 260.

Fig. 6. Trabecula of bone (tibia) lined by osteoblasts. \times about 260.

Figs. 7, 8. From transverse section of tibia. (A. i. in.) Fig. 7 shows new bone growing in periosteum. Fig. 8. Taken from immediately beneath the periosteum.

PLATE III. See pp. 46-7.

Fig. 9. From perpendicular section of skull. (A. i. in.)

Fig. 10. From section of tibia, to show eaten-out border of widened Haversian canal. Oc. 3, obj. 7. \times 200.

Figs. 11, 12. From transverse section of tibia. Fig. 11. At some distance from surface. Fig. 12. From a little way beneath the periosteum. Oc. 3, obj. 4, t. dr. o. \times 87.

Fig. 13. Transverse section of normal tibia. Oc. 3, obj. 4, t. dr. o. \times about 87.

PLATE IV.—Upper and lower ends of femur. See p. 45. (College of Surgeons Museum, No. 395B. Half diameter.)

PLATE V.—Cranium. See p. 44. (College of Surgeons Museum, No. 395A. Real size.)

Fig. 1.



Fig. 2.



Fig. 3.



Fig. 4.

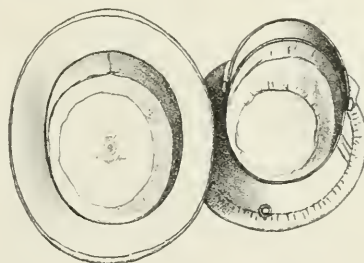


FIG 2

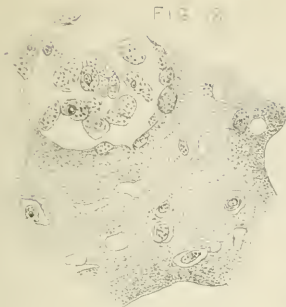


FIG 1



FIG 3

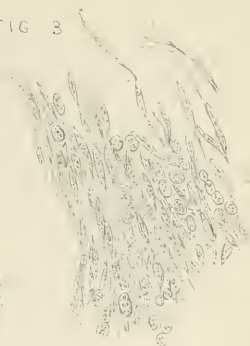


FIG 6

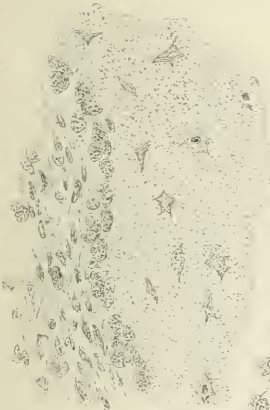


FIG. 4

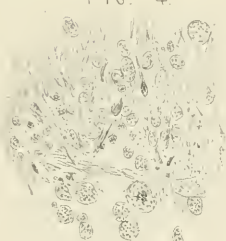


FIG 7

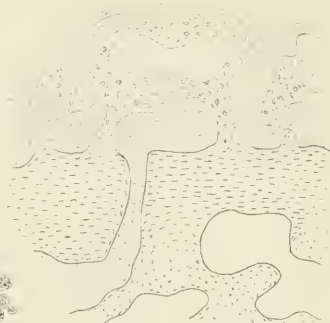


FIG 5

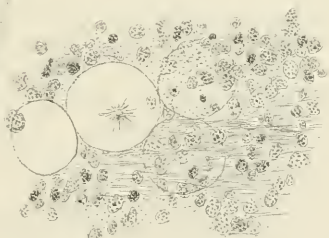


FIG. 8.

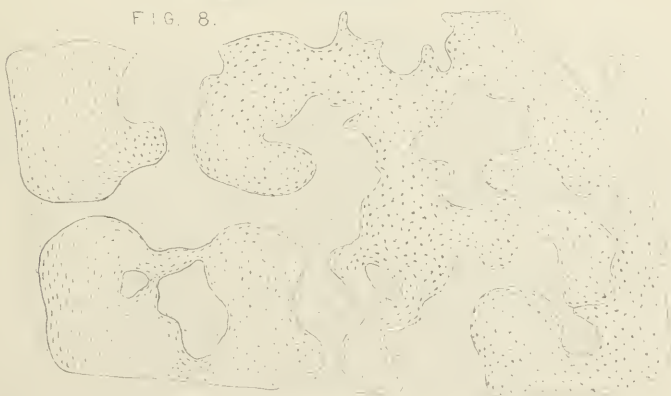


FIG. 9.



FIG. 10.



FIG. 11.

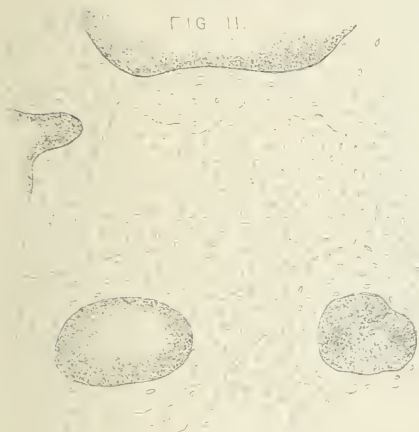
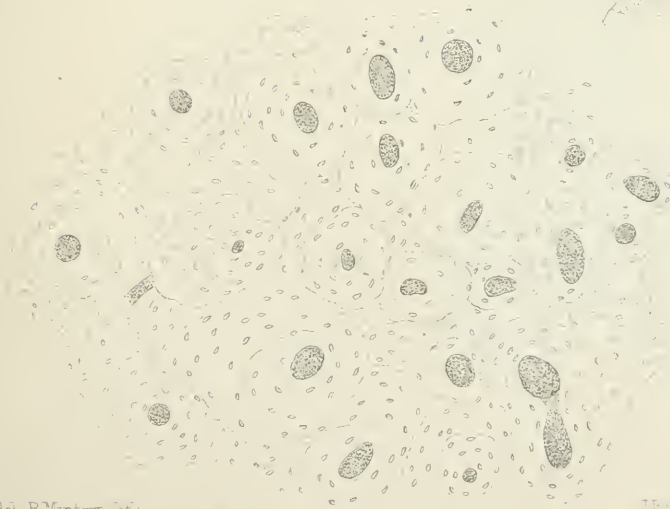


FIG. 12.

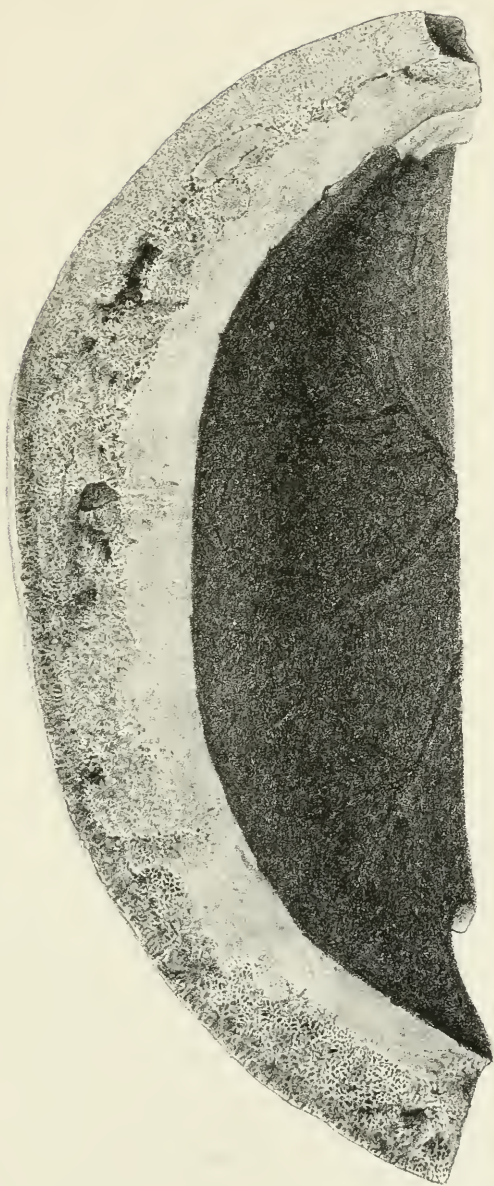


FIG. 13.





Curve of the Femur.



A CASE
OF
COMPLETE ABSENCE OF BOTH THE
UPPER LIMBS

AND OF
FAULTY DEVELOPMENT OF THE RIGHT LOWER LIMB.

BY
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(Received November 21st—Read November 28th, 1876.)

SUCH extreme congenital deformity as that exhibited in this child is so unusual that I venture to think a short account of her will not be uninteresting to the Fellows of this Society. Her singular condition attracted my attention last year when she was brought to the Middlesex Hospital to see an elder sister, then under my care in the accident ward. Observing the interest the child excited her mother allowed me to examine her and to have the accompanying photograph of her taken by Lenthall (see Plate VI). Dr. Lyall, our surgical registrar, obliged me by taking the memorandum which forms the substance of this note.

Mary B—, æt. 4½, apart from the deformities to be described, a well-grown, healthy, pretty, intelligent blonde, is the youngest of four children, none of whom, except

herself, are remarkable for any bodily peculiarity. Her father is a carman, her mother a dressmaker; both are well-formed and healthy.

The child's upper limbs are both completely absent, which gives the upper part of her naked trunk a very angulated, narrow figure. The lateral contours slope vertically, with a slight outward slant from the extreme points of the angular shoulders to a little above the level of the nipples, whence, bending inwards, they fall upon the middle ribs of a well-shaped chest. The clavicles and scapulæ appear well formed. They are of a size corresponding to her stature, and the muscles attached to them act vigorously. Below the projecting acromion no trace of humerus or of any of the distal bones of a limb can be discovered on either side; a cushion of soft tissues fills the angle between the axillary border of the shoulder-blade and the side of the chest. Below the outer end of the right collar-bone, nearly in the level of the vertical semi-diameter of the scapula, is a minute congenital scar, and slightly below and outwards from this is a little dimple. No mark of any kind is discernible on the left shoulder.

When placed upright on her left foot the right limb hangs from the trunk, its foot not reaching the ground by several inches. The thigh is very short and plump; the leg is well shaped, but smaller than the left. The foot has a moderate degree of talipes equino-varus, and the fourth and fifth toes are joined together, the fourth cleft being absent. A very short misshapen femur can be made out, and both bones of the leg are clearly present; contrasted with the femur they seem well developed. The mobility of the thigh upon the pelvis is very great; abduction and circumduction are especially free. She readily puts the thigh through most of the movements of a ball-and-socket joint. The knee-joint is loosely knit; in addition to wobbling hinge movements, the axis of the tibia can be made to rotate upon the lower end of the femur, not only in flexion, but also in extension of the knee joint, and some amount of lateral gliding is also possible.

The left lower limb is perfect. It dexterously performs many offices usually devolving on the hands. The toes are very mobile and prehensile, and so under control, that with the foot she readily grasps and steadily holds even small objects, and executes complex concerted acts, as picking up a comb and arranging her hair, feeding herself with a spoon, and even using a needle. She sometimes makes use of her right (distorted) foot to fix anything she wishes to grasp with the left.

Comparative measurements of the limbs.

| | From antero-superior spina illi to malleolus internus. Inches. | From same point to inner femoral condyle. Inches. | Girth of thigh. Inches. |
|-------------|---|--|----------------------------------|
| Left . . . | 14 | 7 | 11 $\frac{3}{4}$ |
| Right . . . | 8 | — | 12 $\frac{1}{2}$ |

Her mother states that six weeks after conception she met and stopped to look at a beggar in the street who was without arms and had both feet clubbed. This did not make any impression of terror or anxiety upon her, and the circumstance had nearly passed from her memory when recalled by the inquiries of friends.

Interesting as is the vexed question of the influence of maternal emotions upon the embryo, I would suggest that the chief interest of this case lies in another direction. The support it affords to the theory that these and similar defects of the limbs are due to absence, or arrest, or misdirection of development, and not generally, as is very widely thought, to amputation of already formed limbs by their adventitious constriction. The small scars and the little appendages of the present (such as might result from the healing of a wound after removal of part or of an entire member at its junction with the trunk), and the occasional appearance of a furrow upon a limb, suggestive of its having been tightly girt by the umbilical cord or by a band of thickened membrane, are the chief evidence adduced in favour of the hypothesis of intra-uterine amputation. As regards the umbilical cord, however, it is not

easy to understand how it can become so tightly coiled around a limb as to cause this to drop off without itself being, at the same time, so stretched as would greatly lessen the volume of the umbilical arteries and vein, and so seriously prejudice the life of the embryo. Yet, as in this case, well-grown offspring are born at full term with quasi-amputated members. The scars and little appendages adduced in favour of amputation *in utero* admit of a better explanation; they may be, and probably, usually they are, vestiges of members blighted at an early stage of development from some intrinsic defect in the germ, as the author of the 'Catalogue of Monstrosities in the Hunterian Museum' and others have suggested. This is strengthened by their frequent association with other defects of a kind which cannot, from their very nature, owe their origin to any such mechanical constriction as is imagined, viz. deficiency of brain and the like, and redundancy of parts.

The total absence of scar from the child's left shoulder excludes absolutely the notion of amputation. It can only be satisfactorily explained by arrest of development at a very early stage before the projection of a limb-bud beyond the surface of the germ. The scar and dimple on the right shoulder imply that here a limb-bud had begun to grow out, but was blighted in its very inception. The defects in the right lower limb signify a still later disturbance of development. Differentiation of tissues and skeletal segmentation took place, but the growth of the limb, and in particular of the thigh, was stunted, and articulations were never perfected.

The equino-varus may be regarded as a continuance, in a somewhat exaggerated degree, of the normal condition of the foot at birth, which, as Dr. Little has pointed out, has the sole turned slightly inwards. The persistence and increase of this infantile condition may here be due to the foot never having been brought into use for progression in the erect posture.

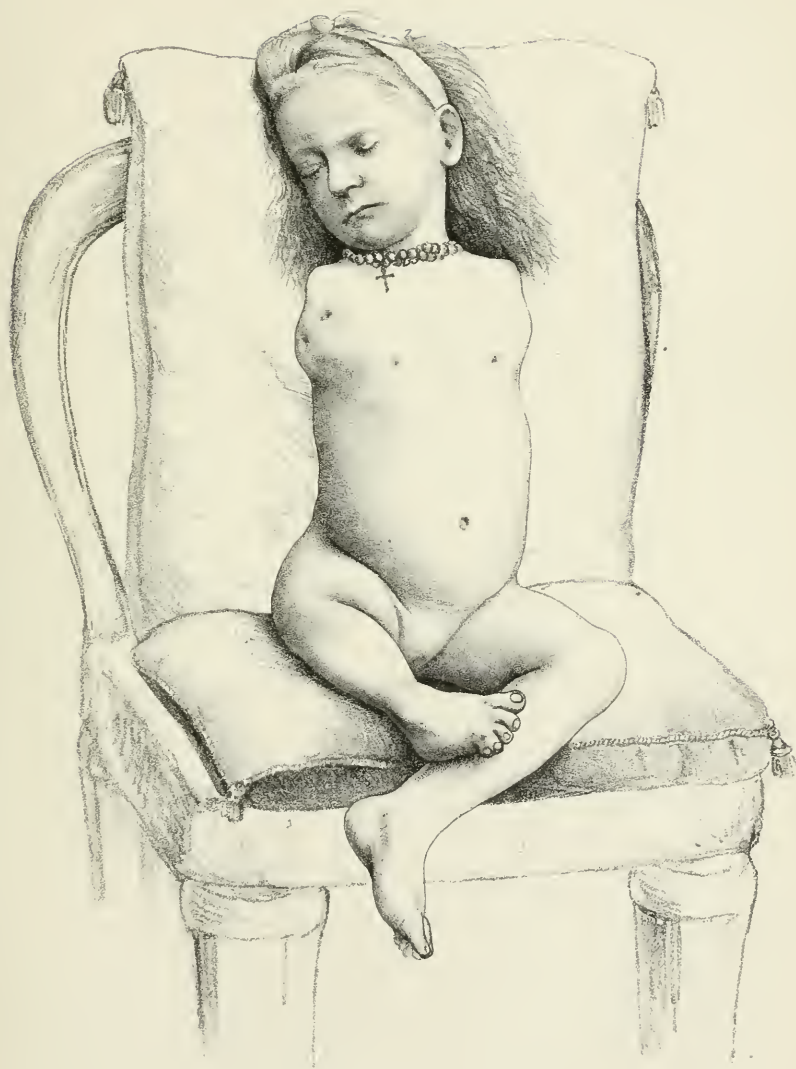
The development of the shoulder-blades and clavicles,

the peripheral segments of the limbs being absent, is normal in *Amphisbænidae* (Blindworms), which have a well-developed shoulder-girdle, but do not possess external limbs.*

* *A case of congenital absence of both arms*, was read February 14th, 1873, at the Clinical Society, by Mr. Thomas Smith.—Thomas C—, æt. 4, a robust, healthy boy. No bands or adhesions found when born, and no vestiges of limbs within the membranes; upper limbs entirely absent. A small dimple on each side upon the prominence of the shoulder, over the position of the glenoid cavity; no projecting stump of an arm and no trace of humerus. The clavicles well developed and articulating normally with the acromion on either side; glenoid cavities empty; free movement of shoulders in every direction; can grasp objects between his cheek and his acromion process; prehensile power of his toes well developed; can pick up a small coin easily.

DESCRIPTION OF PLATE VI.

Case of Complete absence of both the Upper Limbs, &c. (J. W.
HULKE, F.R.S.)



ON THE USE
OF
FLEXIBLE TRACHEOTOMY TUBES.

BY
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FOR SICK CHILDREN.

(Received November 25th—Read November 28th, 1876.)

THE evils which frequently result from the use of silver or other rigid tracheotomy tubes are well known. Pain and irritation, accompanying and following the changing of the tubes, with troublesome bleeding from the granulating surfaces of the wound, are very common, the trouble, however, varying infinitely in different cases ; while ulceration of the trachea is often met with in post-mortem examinations. These evils are, of course, proportionately much more common in children than in adults, and with some forms of tracheotomy tubes than with others ; but they cannot be always avoided.

In a valuable paper on 'Tracheotomy in Children,'* Mr. Howard Marsh remarks, "Ulceration about the wound, or of the trachea around the cannula, is an event which, I believe, is much more common and more formidable than it is generally supposed to be. I have notes of

* 'St. Bartholomew's Hospital Reports,' 1867.

ten cases in which it took place. In four of these it was followed by a fatal hæmorrhage; in a fifth bleeding occurred, but was arrested with the actual cautery; in some of the remaining instances, in which the ulceration was situated at the upper extremity of the wound, coarse vascular granulations arose and obstructed the canal of the trachea, so as to hinder for a long time the removal of the cannula. The importance which the accident derives from its tendency to be fatal is increased by the probability that its occurrence depends almost entirely on the size and construction of the cannula that is employed. The majority of the cases referred to were under the care of those who were biassed by the impression that as large a tube as possible should be introduced, and in six of the cases a Fuller's tube was used throughout.

The ulceration was found, in those of the fatal cases of which I have notes, chiefly in one of two situations, either opposite the lower end of the cannula, or at its upper and posterior curvature." Among the cases, fatal from *various* causes, which are quoted by Mr. Marsh, was one in which the "thyroid cartilage was a good deal eroded;" in another, "around the wound the mucous membrane was found rather largely ulcerated," and "half an inch below the wound was another ulcer about as big as a pea." In another case, "for about three quarters of an inch below the wound of operation, the whole circumference of the trachea was observed to be covered with a dirty slough of the mucous membrane, which being removed, the cartilages beneath were left exposed."

Mr. Bryant remarks,* "The rigid tube, when required to be worn for long, too often excites ulceration by the pressure of its free end on the trachea, which may extend into the innominate artery."

Mr. Durham observes,† "Attention to the size, shape, and general construction of the cannula is of great importance." "Sometimes the cannula is too short, and

* 'The Practice of Surgery,' 2nd edition, vol. ii, p. 31.

† 'System of Surgery,' edited by T. Holmes, 1870, vol. ii, p. 508.

the neck-plate becomes buried in the wound. Sometimes it is too long, and presses against the œsophagus behind, and projects in front to a greater or less extent from the wound, in which case it is very liable to be forced out. The cannulas in ordinary use are so curved that the lower extremity often impinges on the mucous membrane of the anterior wall of the trachea, and sometimes the convexity of the curve comes in contact with the posterior wall. In either case ulceration may be produced sooner or later, and very serious results may follow. Two cases have occurred in Guy's Hospital, in each of which ulceration so produced extended through the anterior wall of the trachea into the innominate artery. Fatal hæmorrhage, of course, ensued. Another instance of the same kind is recorded by Mr. Wood." "Again, abscesses, bronchitis, pneumonia, pyæmia, and other more or less fatal conditions have sometimes resulted, directly or indirectly, from the inflammation or ulceration set up by the cannula."

Many attempts have been made to construct metal tracheotomy tubes in such a manner as to free them from the dangers attendant on their use, and with more or less success. The movable collar, as in French tubes, the ball-and-socket arrangement devised by Mr. Bryant, and, could the workmanship and material be perfect, the lobster-tailed cannula of Mr. Durham, are all improvements on the old-fashioned shapes, while the vulcanite tubes have been found, in many cases, to answer well. But the difficulty has been overcome only in part.

It had long occurred to me that an elastic tracheotomy tube might be constructed which would answer all the purposes of the rigid cannula, and at the same time be free from its disadvantages; but it was only last year that my idea was brought into working shape by my friend Mr. Paley, House-Surgeon to the Evelina Hospital, to whom I am indebted for taking much trouble in superintending the construction of an India-rubber tube, made by Mr. Millikin, of St. Thomas's Street.

This tube was made of ordinary India-rubber, and

answered the purpose for which it was intended very fairly ; but it was evident that a better material would be preferable, and the cannulæ have been since constructed of vulcanised red rubber, a material which is in a high degree elastic, tough, and durable, and remains almost unaltered after long soaking in pus or other like fluids.

The shape of the cannula is that of the ordinary silver tracheotomy-tube. There is no laryngeal opening, but this can be made at any moment, with a sharp knife or scissors, at the part of the tube which seems best for the case in which it is being used. The tube is single, and it has been found hitherto so easy of introduction and withdrawal, or, in other words, one tube can be so easily replaced by another that I have not thought it necessary to devise a combination of inner and outer tubes, as in the case of the silver cannulæ commonly in use. Indeed, the removal and replacement of the single flexible tube can be effected far more easily than a similar change of the innermost of the silver tubes, when two are employed, and with much less discomfort to the patient. Under some circumstances, however, a double tube would be very serviceable ; and I hope to have such a one constructed.

The elastic cannula can be easily introduced, as a rule, without any help in the shape of "guide" or forceps ; but, occasionally, it is found more convenient to employ one or the other. A pair of dressing-forceps has been found to answer well, the tube being compressed near its end ; or forceps may be used like those shown to the Society, which are also of suitable shape for acting, if required, as a trachea-dilator. An elastic catheter, introduced through the tube, so as to act as a guide, has been also found sometimes of service.

Hitherto the elastic cannula has been inserted at periods varying from one or two days to as many weeks after the operation, a silver tube having been previously in use ; but I am disposed to urge that the single tube should not be used earlier than three or four days after operation, not because there would be any risk of contraction and

consequent interference with the patency of the cannula at an early period (for I believe there is no danger of this if the tube be properly constructed), but on account of the disadvantages attendant on the use of all single tubes, while the trachea is still freely movable beneath the soft parts; in other words, when the collapse of the cut edges of the wound, together with a very little want of due relation between them and the incision in the soft parts, may render the reintroduction, especially of a flexible tube, difficult. With a *double* elastic tube this period may, probably, be shortened with advantage, but experience is wanting with respect to it.

The first question which will naturally occur to all is, whether the elastic cannula is capable of withstanding the nipping action of the margins of the recent wound in the trachea, and of those of the wound in the soft parts also, at a subsequent period, when only a fistulous track remains. The answer may, I believe, be given most positively that there is no danger from this cause, and that the tubes may be worn for months without a mark on them to show where they have been gripped; and this, notwithstanding the strong tendency to contraction on the part of the wound. In two cases which have come under my notice, it was impossible, after the removal of the elastic cannula for a few hours, to reintroduce it, and a silver one could be substituted only by using some little force; indeed, in one case, several minutes elapsed before the house-surgeon could complete the introduction of a silver tube. But both before and after this, there was not the slightest difficulty in the introduction of the flexible tubes, when they were being used almost continuously, nor the slightest evidence of obstruction on account of pressure by the edges of the wound.

One well-known disadvantage of rigid tracheotomy tubes lies in the difficulty of knowing beforehand exactly the shape and size which are suitable for any given case, a very slight difference in these respects making a very great difference in the comfort, and sometimes, when the

tube is long worn, in the safety of the patient. This objection is quite removed by the use of flexible tubes, which accommodate themselves readily to different cases. At the same time, if rather too long, they can be shortened with a knife or scissors, according to the requirements of the individual case. Moreover, when two cannulæ are used alternately, one may be made rather shorter than the other; and thus, the point of pressure, so far as the extremity of the tube is concerned, is changed every few hours, an object of some worth, probably, even with flexible tubes, although not to the same extent as in the case of rigid ones.

The advantage of flexible tubes in cases in which, on account of some slight deviation from the middle line of the wound, either in the trachea or soft parts, a rigid tube will not "sit" well, is obvious.

It is, perhaps, scarcely necessary to refer to the greater safety of elastic tubes, when their removal and reintroduction are entrusted to nurses or others not accustomed to surgical manipulation. Practical experience, as will be seen from the appended notes of cases, has shown, too, that there is rarely any corresponding disadvantage with respect to greater difficulty of introduction. It may be well, however, to remind those who may use the flexible tubes, that it is absolutely necessary, during their employment, to have always near at hand either a bivalve silver tube, or, much better, a simple trachea-dilator, of which the kind shown to the Society is perhaps the best, so as to be prepared for any emergency. I have a record at present of four cases in which a flexible tube could not be introduced. In one, the attempt was made at too early a period after the operation, and the silver cannula was therefore replaced. In two others, the difficulty occurred during the period of convalescence, when the elastic tube had been left out for a few hours. After the substitution for a short time of a silver for the elastic tube no further difficulty occurred, and the use of the latter was resumed. With a proper guide or dilator, even in these cases,

probably, the flexible tube might have been reintroduced; but until further experience has been gained, it will be best to have a bivalve silver tube at hand for an emergency. The circumstances of the fourth case, unfortunately fatal, were peculiar, and will be best shown by the appended note from Mr. Clutton. It is not clear that in this case the blame is fairly chargeable at all to the flexible cannula, but if it be so, the case only forms an additional warning of the necessity of having a dilator always at hand.

The elastic tubes are very durable. In one case two of them, used alternately, lasted for six months, and would have lasted longer had they been required. If they have been laid by, however, for a time, they should be roughly handled before use, in order to test their strength and pliability, and no tube should be employed if, with the strongest pressure, there is the slightest tendency on the part of its walls to stick together—an event which can only happen with inferior kinds of red rubber. I have seen it occur only once.

Under certain special circumstances elastic tubing of different kinds has been occasionally employed as a substitute for the ordinary rigid cannula, but, so far as I can discover, India-rubber tracheotomy tubes have never been made hitherto for systematic use. The idea, however, is so obvious, that I cannot doubt it has struck many besides myself, and that the way has been so far prepared for their adoption.

The following are brief notes of some cases in which flexible tracheotomy tubes have been used. I am indebted for the details to Mr. Paley, house-surgeon, and Mr. Andrews, registrar, at the Evelina Hospital, to Mr. Edwards, late house-surgeon at St. Bartholomew's Hospital, and to Mr. H. H. Clutton, resident assistant-surgeon to St. Thomas's Hospital.

I am informed also by Mr. Mason, house-surgeon to the Children's Hospital, Great Ormond Street, that the flexible tubes have been found there to answer well the purpose for which they were constructed.

CASE 1.*—This, the first case in which the flexible tube was employed, was that of a little girl two years old, who was admitted into the Evelina Hospital, October 5th, 1875, under the care of Dr. Frederick Taylor. There was evidence of considerable laryngeal obstruction. No diphtheritic membrane could be seen, and the urine contained no albumen. The symptoms not abating under the use of the steam-tent and other measures, tracheotomy was performed by the house-surgeon, Mr. Paley, on the evening of the day of admission into the hospital.

The case did well, and at the end of a fortnight a flexible tube of ordinary India rubber was substituted for the silver one, and was changed every two days. It was introduced with less pain, was more comfortable, and caused less irritation than the metal tube. The tube was worn for a month, during ten days of which the child was running about the ward. At the end of this time its use was discontinued, and soon afterwards the patient left the hospital well.

CASE 2.—A boy, $3\frac{1}{2}$ years old, was admitted into the Evelina Hospital, under the care of Dr. Baxter, September 5th, 1876, with symptoms of croup. Four days afterwards there was great dyspnœa, and tracheotomy was performed, in the absence of the house-surgeon, by the registrar, Mr. Andrews. On the fifth day after the operation a vulcanised red-rubber tube was substituted for the silver. It could be removed and reinserted with much less difficulty and discomfort to the patient than the innermost of the two silver tubes which had been previously employed, and it seemed to excite less exudation from the operation-wound, and was easily kept clean. The changing of one elastic tube for another could be managed, it was found, with almost complete absence of discomfort to the patient.

On the ninth day after the operation the tube was removed for two hours, and then easily reinserted. On the following day it was again removed, and there being

* Published in the 'Lancet,' December, 1875, by Mr. W. E. Paley.

no recurrence of dyspnœa, the wound was allowed to heal, and soon afterwards the patient left the hospital well.

CASE 3.—A girl, $2\frac{3}{4}$ years old, was admitted into the Evelina Hospital, under the care of Dr. Baxter, September 24th, 1876. She had suffered from sore throat and difficulty of breathing, it was said, for a week. At the time of admission there was marked laryngeal cough, but not much interference with respiration. The fauces were injected, and the tonsils covered with greyish, doubtfully membranous exudation. The breath was offensive.

September 28th.—The child seemed better, but there was a patch of exudation on the uvula. On the evening of this day the breathing became laboured, and with only a temporary improvement on the 29th. Dyspnœa became urgent on the 30th, and tracheotomy was performed by the house-surgeon, Mr. Paley.

October 3rd (third day after operation).—The silver tube was replaced by an elastic one without any difficulty. The latter was not changed for twenty-four hours, and at the end of this time it seemed less foul than is commonly the case with the metal tube at the end of a much shorter period.

During the following week the use of the elastic cannula was continued, and on the tenth day after the operation, the patient having done well, it was left out for six hours. Some dyspnœa, however, then occurred, and an unsuccessful attempt having been made to reintroduce the flexible tube, a silver one was with much difficulty substituted. Two days after this the silver cannula was again replaced by the elastic tube, which was worn at intervals for five days more, when it was discontinued, and no further difficulty of breathing occurring, the child was discharged well October 28th. The ease with which, excepting the occasion referred to, the elastic cannula could be introduced in this case was very marked. It was changed every morning by the sister-in-charge.

CASE 4.—A boy, $4\frac{1}{2}$ years old, was admitted into St. Bartholomew's Hospital, under the care of Dr. Black, February 22nd, 1876, with symptoms of croup. On the following day tracheotomy was performed by the house-surgeon in charge, Mr. Edwards.

February 27th (fourth day after the operation).—An India-rubber tube was substituted for the silver, and changed once every twelve hours. It was found to be less irritating than the silver tube, and did not become clogged so readily. It could also be withdrawn and reintroduced with much more ease than the innermost of the two tubes previously employed.

March 1st (eighth day after operation).—The tube was taken out to-day for about half an hour, but at the end of this time the child began rather suddenly to suffer from dyspnoea, and the wound had so far contracted as to render the reintroduction of the tube difficult. It was managed, however, after a little trouble, by the sister of the ward.

3rd.—The elastic tube was left for sixteen hours without changing. On the following day a laryngeal hole was made in the tube. Up to this date the flexible tube had been occasionally changed for a silver one, but after March 9th two flexible tubes were employed alternately.

15th.—The child can articulate well when the outer opening of the tube is closed by the finger.

From this time onwards the patient did well, but all attempts to dispense with the use of the tube proved unsuccessful. On one occasion, when it had been left out for an hour or two, it was found impossible to reintroduce it, and a silver cannula was, with some little force, substituted. The use of the flexible tubes was, however, resumed a few hours afterwards.

At the beginning of August (five months after the operation) the child was sent to Brighton, and Mr. Langdale, house-surgeon to the Sussex County Hospital, informs me that the elastic tube continued to be worn, and was changed once daily. Several attempts were made, Mr. Langdale adds, "to accustom the boy to do without the

tube, but he suffered from too much spasm of the glottis, until on one warmer day than usual the tube was left out for half an hour, and the length of time was increased every day until the wound became so contracted that the tube could not be replaced at all, and eight weeks after admission (seven months after the operation) the wound closed altogether. The vocal cords seemed perfect, the boy's general health improved, and he left the hospital well."

CASE 5.—A little girl, 5 years old, was admitted into St. Bartholomew's Hospital, under the care of Dr. Black, June 30th, 1876, suffering from croup. On the same day tracheotomy was performed by Mr. Edwards, house-surgeon, and a silver bivalve cannula was introduced. Temperature 103.8° ; pulse 150; respiration 32. In the course of the following day the breathing continued difficult and hurried, and there seemed to be a considerable quantity of mucus, which the child had no power to expel.

July 2nd (second day after operation).—In the course of the morning it was necessary to remove the cannula three times, and on each occasion Mr. Edwards, with polypus-forceps, drew out through the wound several pieces of false membrane. On the same day (thirty-six hours after the operation) an India-rubber tube was substituted for the silver, with marked improvement in the breathing. On the night of July 2nd the breathing became much worse suddenly, and the patient died fifty-four hours after the operation. At the post-mortem examination a piece of false membrane was found stretched across the bifurcation of the trachea.

I am indebted to Mr. H. H. Clutton, resident assistant-surgeon to St. Thomas's Hospital, for the following account of two cases in which the elastic tube has been employed.

"St. Thomas's Hospital; November 26, 1876."

"We have had two cases of tracheotomy in the adult since August, in which the elastic tube was used.

“The first case, a man about 30 years of age, wore a silver bivalve for about a month. It was then changed for the India-rubber tube, as it seemed evident that he would have to wear one for some considerable length of time. The man has uninterruptedly worn this elastic tube for now over two months, taking it out and introducing it himself, with the help of a looking-glass, as easily as he could the silver one. He is now an out-patient, still wearing the tube. He finds it much more comfortable, and would on no account change it for a silver one, as it readily allows him to turn his head in any direction without pain. With cleanliness, too, it keeps perfectly sweet.

“The second was a case of syphilitic laryngitis in a woman about 23 years of age. The elastic tube was introduced about a fortnight after the operation, with very great relief to the patient. No guide, such as a pilot, dilator or forceps, was necessary for its introduction. The lips of the external incision had a tendency to close the wound by the drawing in of the sides during inspiration; but as soon as they were separated by the finger and thumb, one on each side of the wound, the opening in the trachea, which had by that time become firmly adherent to the soft parts, was seen to be patent and fixed, so that the elastic tube could with ease be introduced, contrasting strongly with the condition which is seen for forty-eight hours at least after the operation, in which the trachea is jerked up and down, and requires something stiff and probe-pointed for the easy introduction of a tube.

“The elastic tube was in this case, as in the first, a great comfort to the patient. She could turn and move her head without fear or pain, and the irritating cough which she had had with the former tube was much lessened on the introduction of the elastic one.

A sad sequel occurred to this otherwise successful case. The house-surgeon and myself were suddenly called on Sunday afternoon and found her dead, with the tube lying on the locker. The nurse's account was that she was seized with a fit of coughing, and was seen to throw the tube upon

the table (for she was walking about the ward), and then to become suddenly asphyxiated. I thought at first that the tube had been dragged out by being forcibly bent upon itself, but this was evidently not the case, for the tube was found on the table with the tape attached, so that she must have deliberately untied it. The *possibility* of the elastic tube, although firmly tied in, being capable of removal without untying the tape should be borne in mind, although we have no proof that it would be a likely accident. In this case no blame can be attached to the elastic tube, for it was evidently deliberately untied.

“As regards replacing the tube I think that possibly if the nurse had had a silver tube by the patient’s side she might have introduced it. But it so happened, most unfortunately, that, although there were two nurses and the sister, who were attached to the ward and had introduced the elastic tube for about six weeks, none of them were in the ward at the time of the accident; the patient was walking about the ward and considered quite safe, except at the time of changing the tube, and she had never before attempted to remove the tube herself. The nurse who was in charge of the ward was unaccustomed to the elastic tube and failed to introduce it; it is possible that if she had had a silver one she might have succeeded, but I am quite certain that if one of the nurses who were accustomed to change her elastic tube had been there they would have found no difficulty in reintroducing it. It could be introduced by itself with forceps or with an ordinary pilot.

“In conclusion, I may say that a patient who has once worn the elastic tube would not, I should think, readily allow it to be replaced by the silver one, and that although we have met with this sad accident, I cannot see that the blame ought in any way to be attached to the tube.”

Postscript.—Since this paper was read a case has occurred in which an India-rubber tracheotomy tube, having become brittle, was broken by a patient while in use, and a part

fell into the trachea. The patient was admitted into Guy's Hospital, under the care of Mr. Howse, and the tube was subsequently extracted. The fact indicates the necessity, insisted upon in this paper, of examining the tubes carefully from time to time, and discarding them if they have lost their due elasticity. To prevent, as far as possible, any such occurrence in future, the tubes are now constructed, in accordance with a suggestion kindly made by Mr. Donald Napier, with the addition of a canvas lining between the layers of rubber.

A CASE
OF
LODGMENT OF A TRACHEOTOMY TUBE
IN THE RIGHT BRONCHUS
AND ITS EXTRACTION.

BY
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(Received November 25th—Read November 28th, 1876.)

THE tube in this case was not an old one, which, thinned by chemical action and by friction consequent on long wearing, had broken away from the shield and fallen down the trachea, but a nearly new one, I believe, never previously used, of a pattern which I am informed is now more frequently supplied by London instrument makers than any other. As a warning, which may perhaps save some surgeon the anxiety which the accident gave me, but also and chiefly for any useful hint it may furnish to whom it may fall to have to treat a similar case, I venture to bring this case before the Fellows of this Society.

At 8 p.m. 30th July, Dr. King, in temporary charge of Dr. Greenhow's patients, requested me to see, with him, H. D—, æt. 37, a tall, thin woman, an ironer, who the day before had been admitted into Northumberland ward with laryngitis. Her history showed syphilis, a chronic chest complaint, and indulgence in drink. Difficulty of breath-

ing had come on rapidly four days before. We found her sitting up in bed, unable to lie down, restlessly and impatiently tossing about, speaking in a husky, scarcely audible whisper. Inspirations 36 per minute, shallow, with deep pitting of the infra-clavicular fossæ and of the epigastrium. Pointing to her throat, she begged by signs rather than speech that something might be promptly done to relieve her great distress, and signified her willingness to submit to any measures we advised. Dr. Greenhow, who also saw the case, concurred in the necessity of immediate tracheotomy.

Chloroform was given, the trachea opened, and a Karop's tube inserted. She at once breathed freely and she passed a quiet night, sleeping and occasionally taking milk without difficulty.

For a week nothing occurred which demands particular notice. Her relief was so complete that she spent much of her time reading, interrupted occasionally by cough, which was less annoying by day than during the night. On 7th August, the ninth day, the outer tube was withdrawn for the first time, for the purpose of cleaning it, and, being unprovided with a pilot-plug, some difficulty was experienced in replacing it; for this reason a Durham's, the only other equally long tube at hand, was substituted, the bluntly-conical end of its pilot-handle making the introduction easy. She found this tube, though externally of nearly similar size to Karop's, much less comfortable; the inner tube choked sooner and it required to be more frequently changed.

Three days later (August 10th), at 5.30 p.m., whilst the inner tube was being replaced after cleaning, the outer tube slipped through the collar and shield, and she felt it slide from the wound deeply into her chest.

The accident greatly alarmed her, and instantly provoked violent cough. Within a few minutes of its occurrence several attempts were made by the house-surgeon, Mr. Karop, to seize the tube with slender long forceps, but he could not feel it, though the forceps were very

deeply passed up to their rings. He then inverted the patient, and shook her, and smartly slapped her back, but failed by these measures to dislodge the tube. Half an hour later I myself repeated these manœuvres, unsuccessfully. Neither with forceps, nor with a loop of copper wire could I get metallic contact, perhaps because of the difficulty of manipulating with the former, and because the latter was too flexible for its accurate direction.

The paroxysm of cough had ceased before I saw her, and fearing to inflict more injury by prolonging my efforts, I decided, under the circumstances, to defer further direct interference. Trousseau's largest tube was inserted, and this she found very comfortable—much more so, she said, than the other, Durham's. She now breathed tranquilly without effort. She felt pain at the right of the lower part of the sternum, at a spot to which she signed that she had felt the tube glide down.

No indication of the presence of the tube, except a very slight relative weakness of the respiratory sounds on the right side, was discovered, notwithstanding repeated careful examination of the chest.

11th August.—She had slept fairly well, though cough was sometimes troublesome. She says she is comfortable, but her neck feels a little stiff and her throat sore when she swallows. The pain at the lower part of the sternum continues. P. 82, T. 98·9°.

12th.—Passed a restless night, harassed by cough, sputa copious. Has now pain in the left side on deep breathing. Respiration in left base feeble and percussion note impaired here; no friction or crepitation. At 7.40 a.m. she had a sensation as if the tube moved upwards to the neck, but the house-surgeon who was instantly called, explored the trachea with a forceps without feeling it. In the evening she had pain in the left shoulder. P. 96. T. 90·8° F.

13th. (fourth day).—Cough more troublesome. She feels weak and very faint, and she has a sensation of tightness across the chest. Tubular breathing and moist crepita-

tion over the base of the left lung. At 7 a.m. T. 101·8° and at 10 a.m. T. 103·4 P. 114.

At 11 a.m. she had a severe paroxysm of cough, which expelled a large quantity of mucus; thinking that this might have been provoked by a movement of the tube the house-surgeon again examined the trachea with a forceps, but with no better success.

At 1 p.m. T. 103°, P. 120. The tube was evidently provoking inflammation. Its immediate removal offered the only prospect of rescuing her. Chloroform was therefore given, her head allowed to drop back, and neck stretched out. Trousseau's tube was withdrawn, and a long piece of German silver wire, one end of which was bent into a blunt hook, about one eighth inch long, and the wire again bent at about one and a half above this at an angle, roughly estimated at that which the right bronchus and trachea include, was passed through the wound down the trachea. The other end of the wire was bent in a large loop, the plane of which coincided with that of the tracheal end of the wire beyond the angle. This coincidence told the direction of the hook, and so enabled me to keep it in contact with the right side of the trachea and to guide it into the neighbouring bronchus. When the hook had been passed so deeply that I judged it must have reached the end of the trachea I distinctly felt it touch the tube; continuing to slide the hook onwards until from the known length of the tube, I thought it must be beyond it, and then withdrawing it, I was sensible, from the resistance, that the tube was hooked and was moving upwards. When it had almost reach the tracheal wound the hook became disengaged, but the tube was here easily seized with a forceps and taken out. Trousseau's tube was reinserted. At 9 p.m. her temperature had fallen to 99·4° Fahr., and her pulse to 96 per minute.

14th.—Next day, she felt, she said, very well. Her cough was less troublesome. She breathed with perfect ease, and had lost all pain and tightness in the chest.

From the time of the extraction of the tube all threaten-

ing symptoms disappeared, and on my return from my autumn vacation I found she had been discharged from the hospital, 15th September, convalescent, wearing, however, a tube the use of which it was thought she could not safely discontinue.

I did not see her again till 26th October, at which time her voice, when she temporarily stopped her tube, was so strong and clear that I thought she ought to try to do without it, and I took her into the hospital for a couple of days, in order that if dyspnoea followed the closure of the wound this might be immediately dilated, and a tube reinserted. In forty-eight hours the wound had so contracted that it would scarcely admit a small probe. She breathed easily and she spoke well. She continues, I believe, free from any tracheal embarrassment.

Epicrisis.—How the set-screw which, in Durham's tube, regulates the length of the part of the tube behind the collar, and secures it to this, became loosened—whether by overwinding or reversing—could not be learned, for neither nurse or patient were aware of having turned it. The screw ceasing to act, the collar no longer tightly embraced the outer tube, and, this not having a flange, there was nothing to prevent it slipping quite through the collar and being drawn down the trachea. This defect in the construction of the tube arises from the abruptness of its curve, which prevents the workman drawing on the collar over the tracheal end of the tube, and obliges him to pass it over the front or outer end, which for this purpose is designedly left without any projecting flange or out-turned shoulder. A few makers remedy this by subjecting the outer end of the tube, after the collar has been put on, to a strong pressure which slightly forces out its outer edge, but the projection thus formed is so very slight that a little wear would remove its efficiency. Most makers, however, do not adopt this precaution, but they leave the outer lip of the front end of the tube perfectly flush with the part behind it. This dangerous defect could be easily removed by having the

lip well shouldered. But Durham's tube has an inherent defect which, I think, outweighs any advantages of curve and power of altering its length, and which makes it inferior, I think, to many other tubes, particularly to Trousseau's. All must agree that in bronchotomy the greatest freedom of breathing is, so far as concerns the tube, attainable only by the insertion of the largest tube which the trachea will contain without risk of injury to its mucosa from undue pressure; and, also, that the efficient lumen of a double tube is that of its inner cylinder at its narrowest part. Now, the inner cylinder of Durham's tube has a jointed composite structure, which necessitates a much greater thickness of metal, reducing in a corresponding degree the efficient lumen. A comparison of a Durham's with a Trousseau's tube, their outer cylinders having the same diameter, will show a very appreciable excess of sectional area in favour of the latter. Again, one of the chief troubles in the first days after bronchotomy is the clogging of the inner tube by the accumulation and inspissation of mucus, requiring its frequent removal for cleaning. The longer the inner tube, and the smoother its surface, the less often will this be necessary. Trousseau's tube has these merits, but the smaller lumen of Durham's, and particularly its jointed structure, makes this more easily clogged, for each joint when the cylinder is flexed, as it is *in situ*, presents to the lumen a projecting ridge on which the mucus may lodge. I do not advance these as theoretical objections, but from observation.

The difficulties attending the extraction of a foreign body from the lower part of the air-passages are only fully estimated when they actually confront us. The most useful instrument in each case will principally depend on the nature of the foreign body. Forceps, to be really useful, should be light enough to serve as a sound, and also sufficiently flexible to allow them to be bent to any desired curve. Such forceps, made of silver, Gross figured at p. 252 in his exhaustive treatise 'On Foreign Bodies in the Air-passages,' published at Philadelphia in

1854. From inquiries I have made they appear to be unknown to London instrument makers, whose forceps want the lightness and flexibility essential for adaptation to particular cases and for delicate manipulation. A long loop of very flexible wire has been used, I have been informed, with success, for the extraction of a worn-out tracheotomy tube from the trachea; but in this case it failed me, and I imagine that it will often do so owing to the inherent impossibility of imparting to it a definite direction when deeply out of sight in the trachea. For the suggestion of the particular sort of wire and hook, which here did me good service, I am indebted to Messrs. Weiss, who on a former occasion had made for me several loop-hooks of different sizes and curves for the extraction of a sixpence from the larynx, lodged in such a position that no forceps could grasp it. The case is recorded in our 'Transactions' (vol. xlviii, p. 201).

The qualities of the German-silver wire, in particular its flexibility, combined with sufficient stiffness—the latter increased by its angulated sectional figure—made it very superior to the cylindrical wires of brass, iron, or copper, I had previously tried.

The question may have occurred to some why I presumed the tube to be lodged in the right bronchus. Independently of the known greater frequency of foreign bodies lodging in the bronchus, the woman's sensations as the tube slipped down, and the fixed pain at the right of the sternum during the first two days, together with the immediate slight weakness of breathing on this side, weighed with me more than the later indications of inflammation on the left side which supervened on the fourth day. That the physical signs of the presence of the foreign body in particular weakness of breathing sounds on the right side, were not more strongly marked, was doubtless due to its tubular shape, which allowed air freely to traverse it.

Although the trachea was more than usually deep,

owing to much thickening of the overlying structures, the lower thyroid veins were turgid; the bleeding so often very embarrassing was here insignificant. This was owing to the adoption of a little manoeuvre which I learned from a German treatise on 'Tracheotomy' some three or four years ago, but the reference to which I have, unfortunately, mislaid. The author points out that the venous plexus overlying the trachea is enclosed between two folds of the deep fascia which pass upwards to the larynx. The veins also are much smaller above than lower down. If this fascia is fixed by pressing the nail of the left forefinger against the front of the cricoid cartilage, and a small cut be made upon this immediately above the nail, in a direction transverse to the axis of the trachea, both these folds of fascia will be divided. If now the lower lip of the cut fascia be seized with a forceps the fascia below, enclosing the veins (and isthmus of thyroid gland, if necessary), may be easily peeled down with the thin handle of the scalpel, exposing the front of the trachea by an almost bloodless dissection. The author reported that he had tried this in many cases of tracheotomy in children and in adults, and already from my own more limited experience I can speak strongly in its favour. I cannot learn that it is generally known here.

Since this paper was read my attention has been called to the record of a case in which Mr. Davy removed, with a hooked probe a broken tracheotomy tube from the trachea of a patient in the Westminster Hospital ('Brit. Med. Journ.,' 1876, vol. ii, p. 47.)—J. W. H.

ON A CASE
IN WHICH A
FLEXIBLE INDIA-RUBBER TRACHEOTOMY
TUBE WAS REMOVED FROM THE
RIGHT BRONCHUS.

BY
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SICK CHILDREN.

Received March 12th—Read March 27th, 1877.

J. S—, æt. 43, was admitted into Guy's Hospital on December 25th, 1876. He gave us the following history. He had been in Guy's in September, 1866, under the care of Mr. Bryant, suffering from difficulty of breathing due to very extensive syphilitic ulceration about the larynx. Tracheotomy was successfully performed and he was sent out, wearing an ordinary double silver tube. He says that he continued to wear this for four or five years, occasionally taking it out to clean it. After this time it was worn much more irregularly, sometimes being left off for days or weeks. He confesses that these periods generally corresponded with times of commercial depression, when the tube used to find its way to the pawnbrokers', being redeemed again when money became more abundant. Once or twice in the ten years which

elapsed between 1866 and 1876, he had been readmitted into the hospital, and had had a fresh tube given him to replace the old worn-out one.

On October 21st, 1876, after a longer period than usual during which he had not worn the tube, he found it impossible to reintroduce it, and came again to the hospital for relief. Mr. Bryant enlarged the aperture into the trachea, and inserted at first a metallic tube, changing it a few days afterwards for one of the new india-rubber ones.

On November 3rd he left the hospital, having learnt the management of the new tube.

Everything went on satisfactorily until the morning of Christmas Day (*i. e.* seven weeks after commencing the use of the new tube), when in pulling it out to clean it, he felt something give way. He continued to pull rather harder, when the tube tore through near the shield, and then in a fit of coughing he felt the tube recede deeply into the trachea. He presented himself immediately for admission.

At 1.30 p.m. chloroform was given and a bent probe was introduced through the opening into the windpipe. This detected a foreign body lying about the bifurcation of the trachea. Loops of wire of various forms and size were at first tried, with the hope of catching the tube, but unsuccessfully. (We had none of the particular form of wire recommended by Mr. Hulke in his previous communication to this Society, and as it was Christmas Day, there was no hope of obtaining any.) After many futile efforts in this direction, the opening into the trachea was enlarged, and forceps of different size and shape were introduced. The left bronchus was now felt almost completely clear of obstruction, while the right evidently contained the tube. Our efforts were therefore directed to this side, and after some time and trouble, the tube was seized and brought to the surface, one blade of the forceps having fortunately passed into the mouth of the tube and the other blade externally, so that

they grasped it when closed. A silver tube was at first introduced; this was changed for a vulcanite one next day, and shortly afterwards the patient left the hospital, seemingly very little the worse for his accident.

On examining the tube (marked A in the specimens sent round) at the point where it had separated, it was found, when in the moist condition, in a very rotten state. It was quite easy to pick off small pieces of the rubber from the torn ends with the gentlest possible traction. A few hours later, when the tube had got dry, it was in a much more firmly coherent state. The reason of this change is not easy to understand, unless the tracheal mucus had had in this case a pernicious action on the rubber. The separation had taken place about a quarter of an inch from the shield, and it seems clear that the way in which it occurred was this. The tube had not been withdrawn for two or three days, so that a certain amount of contraction of the newly formed cicatricial tissue about the wound had taken place, causing the skin very tightly to embrace the tube. In pulling at it the first effect of the traction was to elongate that part outside the skin, hence to diminish the calibre of this portion of the tube, and consequently its strength. It would follow naturally from this that the tube would break at the stretched part. Hence the very elasticity of the tube would be an element of danger in the case.

It has been objected to this view that it would be impossible for newly-formed cicatricial tissue so to contract upon a tube as to hold it so tightly as this view implies. The analogy of a bougie or catheter in the urethra undoubtedly would lead one to suppose that the constant presence of such an instrument would tend rather to *dilatation* than to *contraction* of the surrounding tissue. It must, however, be remembered that the two cases are essentially dissimilar—that in the one case we are dealing with an epithelium-covered surface, and in

the other with a simple contracting cicatrix. Moreover, this was a syphilitic patient, where the tendency to vigorous contraction of the newly-formed tissue would be great. In the tube marked c (one of the first rubber tubes used at the Evelina Hospital), the contraction of the cicatricial tissue has had the effect of altering permanently the outline of the tube, if not absolutely diminishing its calibre. This may be well seen by holding it so as to examine it in profile.

Moreover, the size of the tracheal sinus, as we examined it before the operation, was altogether disproportionately small when compared with the size of the tube, so that it seemed absolutely impossible that such a tube could have gone through it. These facts seem to prove definitely the power of the contracting tissue in this case.

(A second tube in a very similar condition to the first as far as regards rottenness is among the specimens, marked b. It is from a child at the Evelina Hospital, and had been worn little more than a fortnight.)

It seems, therefore, quite clear that these tubes, composed of simple red rubber, as originally described, are not safe articles to trust a patient with outside a hospital. Influenced by these considerations I wrote to the manufacturer and asked him whether he could not construct one with a piece of canvas running from the shield down either side of the tube. This has since been done, and the tube (marked d) is constructed on this plan. It is *inelastic*, though flexible, and hence avoids the above-described danger. Being flexible, however, it answers all the purposes for which these tubes were first devised, *i. e.* it prevents all risk of the canula eating its way into the tracheal wall, and thus causing death from hæmorrhage. Nevertheless, even with this amended tube, the patient ought to be cautioned that when he sees cracks in the rubber, he ought to get a new tube, otherwise the mucus will gain access to the canvas and will gradually rot it.

Another precaution which should be taken with rubber

tubes, but which is very generally neglected, is to use glycerine as a lubricating agent instead of oil. The latter, when long in contact with even the best forms of rubber, is certain to rot it, while the former has little or no effect upon it, or even if it had, would from its solubility be washed or dissolved away by the natural fluids of the tissues.

A CASE
IN WHICH
A SILVER TRACHEOTOMY TUBE WAS
REMOVED FROM THE LEFT
BRONCHUS,
WHERE IT HAD BEEN LODGED SEVEN WEEKS.

BY
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(COMMUNICATED BY J. W. HULKE, F.R.S., *Hon. Sec.*)

Received March 31st—Read May 8th, 1877.

C. B—, æt. 56, had worn a tracheotomy tube for fifteen years, during which time it appears the tube had not been once changed. He was a free drinker, and the operation was originally performed for an injury to the larynx caused by throttling.

On the 28th of December, 1876, he was in the act of replacing the inner tube, after having cleaned it, when the outer tube broke away from the shield and dropped into the trachea. This was followed by a severe fit of coughing, which after a time subsided. He then sought advice at a metropolitan hospital, into which he was admitted, but owing to the slight urgency of the symptoms present, his story seems scarcely to have been cre-

dited. The trachea was probed and a new silver tube introduced, but nothing further was done, and he left the hospital after remaining an in-patient for a week.

He came to Guy's Hospital on January 5th, 1877. At the time he was breathing comfortably, but he stated that he was liable to violent attacks of coughing and dyspnoea, and that these were especially excited by turning on his right side. He consented to be examined under chloroform on condition that he might return home the same day.

Chloroform having been given, Mr. Lucas, in the absence of Mr. Bryant, enlarged the opening and examined the trachea with a long probe. After some trouble the tube was felt, and on one occasion Mr. Durham in using the probe caused a distinct metallic sound, audible to those around.

Inversion of the body was tried, and various forceps were introduced into the trachea, with a view of seizing the tube, but all attempts to secure it proved fruitless. There was a remarkable absence of signs of tracheal or bronchial irritation following this attempt to remove the foreign body, and the man refusing to have anything more done left the hospital two days after.

He presented himself again on January 29th, saying that he had been unable to get any rest at night, or to do any work since he left the hospital. His lips were blue, and he looked more ill and sickly than when last seen. His complaint was chiefly against the tube, which he thought too small. He was examined by Dr. Pye-Smith, who found that there was well-marked dulness at the base of the left lung and absence of respiratory murmur. There was also a diminution in the vocal fremitus, but the heart was not displaced. There was diminution of the respiratory murmur throughout the left lung and also somewhat deficient resonance. There was very little cough and only occasional râles. Dr. Hilton Fagge, who also examined him, thought that there was an intermittent rhonchus over the left bronchus, which

pointed to the situation of the foreign body. The patient was unable to lie on his right side.

A larger tube was inserted, through which the patient said he could breathe much more freely, and, refusing to have anything more done he left the hospital a second time on February 2nd.

He was admitted for the third time on February 16th. He looked ill and worn, and was evidently thinner. The physical signs remained much as before, but there was more general rhonchus and the sputum was distinctly foetid. He stated that he had not left his bed during the time he had been at home, and that he had been troubled with severe cough and pain on the left side. He now consented to be operated upon; chloroform was accordingly given, and the opening in the trachea enlarged. On introducing a probe, a metallic body could be distinctly felt apparently in the left bronchus. Acting upon the experience of Mr. Hulke's case, Mr. Lucas introduced a soft copper wire bent at the extremity into a short well-rounded hook. With this the tube was caught, but it was found impossible to displace it, and further (owing probably to the wire having passed on the side on which the tracheotomy tube lay in contact with the trachea) it became impracticable to disentangle the hook. The wire being of very soft copper the hook was unbent by traction, and on being withdrawn it carried with it a blood clot between four and five inches in length, and of the thickness of one's thumb. After this the breathing, which had become a source of great anxiety, was much relieved. Two or three different forceps were now tried in succession, and the tube was eventually seized by a pair of slightly curved forceps opening laterally, one blade of which had passed into the interior of the tube, whilst the other grasped it from the outside. Seized in this way the tube was withdrawn with the greatest ease.

A red-rubber tube was now introduced, but it showed a tendency to collapse, so that the air in passing caused an unpleasant noise like blowing through a trumpet. As

the irritation subsided the noise ceased, and it was decided to give the tube a trial. Three or four hours later, however, the patient was found in great distress. The tube was partially collapsed and choked with mucus; air during inspiration was sucked in by the side of the tube, and at every expiration some air was driven into the cellular tissue of the neck. Both sides of the neck above the wound were distended by air to the level of the jaw. The substitution of a metallic for the rubber tube gave the patient immediate relief, and he soon after fell into a comfortable sleep.

No rise of temperature followed the operation. There was some bronchial expectoration, which gradually subsided, as did also the emphysema of the neck. More air could be heard entering the left lung after the removal of the tube, but the dulness at the base remained as before. At the end of ten days he was able to sit up and to walk about the ward.

It had been noticed when he first came into the hospital that there was some pitting of his back, when a stethoscope was applied; but owing to attention being directed towards removing the foreign body, and also owing to the short time that he remained in the hospital on the two previous occasions, no examination of his urine was made till after the removal of the tube. The urine was now found to be loaded with albumen. There was also some œdema of the ankles after standing or walking about and slight general anasarca. From this he said he had suffered more or less for about two years, but that it generally disappeared after the use of strong aperient medicine. Dr. Pye-Smith also detected a systolic aortic bruit, which he thought due probably to an atheromatous condition of the aorta. The patient was now freely purged with jalap, and progressed well till March 9th (three weeks after the operation), when he caught cold and began to expectorate a considerable quantity of greenish mucus. He was placed in an atmosphere of steam, and given a mixture containing squill and senega,

from which he derived considerable benefit, but he was still troubled with a good deal of bronchial expectoration when he left the hospital on March 16th.

The tube, which was thickly coated with black sulphide, was found to have broken away from the shield. It showed at the upper end on its convex side the scratches made by forceps in attempts to remove it. On the concave side were marks made by the passage of the wire.

Remarks.—I wish to draw attention to the following points.

I. That in a person who has worn a tracheotomy tube for many years, a foreign body may become lodged in a bronchus without causing any great difficulty in breathing, and without exciting any grave symptoms of irritation, for a considerable period.

II. That instruments may be freely introduced into the trachea in such cases without any fear of exciting serious bronchial inflammation.

III. That if a wire be used as a hook it is very important that it should be of soft metal, lest it should become immovably fixed.

The difficulties experienced in removing the tube were due in a great measure to a lack of proper instruments, all the curved forceps at hand being throat forceps, which were not only ill-adapted for use in the trachea, by reason of their improper curves, but also by their great size, which prevented the blades from being freely opened when inserted into the trachea. Mr. Millikin, of 3, St. Thomas's Street, has since made for me a forceps well adapted for removing foreign bodies from the trachea or bronchi. The blades are light and narrow, being about the length and width of those used for the urethra. The forceps is slightly curved throughout, but near the extremity of the handle the curve is suddenly increased, the object of the increased curve at this part being to allow the operator to work beneath the projection of the chin,

The handles cross slightly, so that the blades may be opened to a considerable distance by a slight separation of the handles.

I have tested this forceps on the dead subject, and have found that with its aid a silver tracheotomy tube may be caught and removed with ease from either bronchus.

The following were exhibited at the Meeting.

- (1.) Silver tracheotomy tube removed.
- (2.) New forceps.
- (3.) Drawing representing the position the tube is believed to have occupied in the left bronchus.

A CASE
OF
GENERAL TELEANGIECTASIS,
MOST DEVELOPED IN THE LEFT HALF OF THE BODY,
ASSOCIATED WITH
ABNORMALITIES OF THE LARGE BLOOD-VESSELS
OF THE LEFT LOWER LIMB AND IN
THE NECK.

BY
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OPHTHALMIC HOSPITAL, MOORFIELDS.

(Received November 21st—Read December 12th, 1876.)

THIS case is so remarkable for the great extent of the nævoid disorder, and for the unexpected abnormalities in some of the largest blood-vessels found at the inspection of the body, that I venture to hope it may not be unworthy of the notice of this Society.

In 1866, when I was assistant-surgeon to the Middlesex Hospital, E. L—, a puny infant, only a few weeks old, was brought to the out-patient room with intertrigo in the groins and in the crease between the thighs and vulva. A few small nævoid specks caught my attention; the mother seemed hardly to have remarked their presence. Under simple treatment the intertrigo disappeared,

and the child passed from my notice until December, 1870, four and a half years later, when her mother brought her on account of painfulness of the left leg. This leg and thigh were swollen and—as also the left half of the trunk, the left side of the head, and left arm—were mottled with dull red nævoid spots, mostly of the flat kind, popularly known as port-wine stains.

Over the chest, arm, thigh, and leg, they were small and scattered, but on the foot and on the face the spots ran together in larger blotches. The intervals of clear skin between the spots and blotches were traversed by dilated veinlets. In the left groin and upon the mons the nævoid tissue formed a prominent purplish spongy mass, the coarsely granulated surface of which was covered by an extremely thin epidermis. In the neighbourhood of this the greatest distension of the cutaneous veinlets was observed. Scattered over the right side of the body were a few small and inconspicuous nævoid marks, similar to those on the left half. In all its dimensions the left lower limb was considerably larger than the right. The child was fractious, she evidently suffered. The mother said that in cold weather she became blue and suffered more, the left leg becoming very painful. She was passionate and less intelligent than most children of her age, and not always cleanly.

Observing the excessive vascularity of the left lower limb I concluded, hastily as it proved, that it must be warmer than the right, and tested with the hand this really appeared so to myself and also to my class, but a careful thermometric examination proved the opposite:

| | Temp. ham. | Temp. groin. | Temp. groin.* |
|-------|------------|--------------|---------------|
| Left | 95·8° F. | 96·7° F. | 99·3° F. |
| Right | 96·9° | 97·7° | 101·4°. |

* A second observation during a feverish attack. Kew certificated thermometers were used. They were placed in contact with the skin, covered with a layer of cotton wool, and retained during several minutes with a bandage.

She left the hospital towards the end of the month, and was readmitted on 3rd January following (1871), having the day before lost blood to a large extent, the mother thought, from the bowel. A similar loss had happened, she said, before this. The anus and rectum were carefully examined, with a negative result; no piles, nor polypus, or diffuse excessive vascularity, was found here. This and the absence of other evidence of bowel disorder led me to think that the mother might have been mistaken as to the source of the bleeding.

In the summer (1870) she had a large loss of blood, the mother thought, from the vulva, and early in December following a very copious hæmorrhage happened in the night; the blood wetted her nightdress as high as the collar, and ran through the mattress. Two days after this I readmitted her into Queen Ward. No undue vascularity of the vulva or vagina was then apparent. The mass of nævoid tissue upon the mons was larger and more spongy, and minute scabs of dried blood were noticed upon some of the little projections which the bunches of its distended vessels formed upon its surface.

18th December.—A fresh hæmorrhage occurred. It was quickly stopped by ice. The house-surgeon clearly traced this to rupture of small bunches of this large spongy mass. The blood trickling down between the child's thighs might easily, when it had ceased to flow, have been thought to have proceeded from the vulva. The general nævoid condition of the surface had made great advance, still decidedly preponderating in the left half of the body, and the cutaneous veinlets in the spaces between the nævoid patches were more numerous and also more distended. Upon the belly and also on the back were several veins of the size of a quill. The disproportional enlargement of the left lower extremity was greater. The nævoid blotches had become almost confluent upon the entire surface of the thigh and leg. The limb was cold, and the nails very blue. A curved ulcer, one inch wide, encircled two thirds of the girth of the leg. The

child was in much pain, very fretful, and feverish ; and in this and several subsequent feverish attacks she had obstinate and frequent retching. The hæmorrhage on this occasion (18th December) did not amount to more than a few drachms. It was quickly stopped with ice. The ulcer healed slowly.

On February 5th, when she went home, the following measurements were taken :

| | From ant.-sup. sp. ilii to malleol. int. Inches. | From same point to patella. Inches. | Girth of thigh. Inches. |
|-------|--|---|-------------------------------|
| Right | $16\frac{1}{8}$ | $9\frac{5}{8}$ | $10\frac{1}{8}$. |
| Left | $17\frac{3}{8}$ | about 1 more | $11\frac{5}{8}$. |

On 15th April (1871), it was noted that the nævoid mass on the mons had overspread the cutaneous surface of the left labium majus, and the general nævoid condition of the whole surface of the body had made great progress. The internal mammary and epigastric veins and their tributaries were very conspicuous.

In May the mother reported another hæmorrhage, as she thought, from the genitals.

October 7th, 1872.—She was readmitted with glandular abscesses in both groins. One had already burst, the other was opened with potassa fusa. They closed very slowly.

25th August, 1874.—She was admitted with erysipelas of the left lower limb, and several small superficial ulcers in the thigh and leg. She was discharged convalescent 18th September. Comparative measurements of the lower limbs showed :

| | Ant.-sup. sp. ilii to malleol. ext. | Girth of thigh. | Girth of knee. | Girth of calf. | Length of foot. |
|-------|--|--------------------|-------------------|-------------------|--------------------|
| Left | $22\frac{3}{4}$ | 11 | $8\frac{3}{4}$ | $8\frac{1}{4}$ | $6\frac{3}{8}$. |
| Right | $20\frac{1}{2}$ | $9\frac{1}{2}$ | $7\frac{3}{4}$ | $6\frac{1}{4}$ | $5\frac{7}{8}$. |

9th August, 1875.—She was readmitted with intense vascular engorgement of the left limb ; the thigh and leg were both intensely purplish red ; they were dappled with

dots and arborescent brushes of capillary hæmorrhage, and extremely swollen, and very painful. Considerable fever and troublesome retching attended this inflammatory attack. Three days later several of the infarcted patches of the skin were gangrenous. The eschars were very slowly thrown off. The sores had little aptitude for healing, and they frequently became filmed with thin grey pellicular sloughs.

By the beginning of October their condition had much improved, but on the 10th of this month a fresh erysipeloid attack undid the repair and greatly weakened her.

By the 28th the erysipelas had passed off, leaving the ulcers pale and indisposed to cicatrise.

4th November.—Erysipelas again attacked the limb, which became extremely swollen, intensely congested, and blebbed. As in the former attacks she retched frequently. Her temperature ranged between 102° and 104° F. Screaming delirium soon set in, and death took place about twenty-four hours from the beginning of the attack.

The post-mortem inspection of the body, obtained with difficulty, was in many respects less perfect than could be desired. Dr. Sidney Coupland, who made it, found that the abdominal aorta underwent a more than usual diminution below the detachment of its cœliac and sup. mesenteric branches. The arteria carotis sinistra and art. iliaca comm., ext. and int. sinistra, were slightly smaller than the corresponding vessels of the right side. The vena iliaca dextra was of normal size, but the left common iliac vein was not larger than the venule accompanying the middle sacral artery, and the external iliac and the femoral vein were replaced by a plexus of veinlets, which with others corresponding to the branches of the art. iliaca int. formed a rete mirabile intercalated between the cava infr., ven. iliac. comm. sinistra above, and the pelvis and lower limb below. The left internal jugular vein was less than half the size of the right. A chain of swollen lymphatic glands accompanied the aorta and

iliac arteries, especially on the left side, where they reached along the femoral artery into the thigh. The bulk of this limb was immensely increased, as will be seen from the subjoined measurements.

| | Girth at top of thigh. | Girth at upper border of patella. | Length from ant. sup. iliac sp. to int. condyle of femur. | From int. fem. condyle to base of 1st metatarsal. |
|-------|------------------------|-----------------------------------|---|---|
| | Inches. | Inches. | Inches. | Inches. |
| Left | 16½ | 10¾ | 11½ | 12½. |
| Right | 10¾ | 7 | 10½ | 11¾. |

Cuts into the swollen limb showed it to be very œdematous, and that the nævoid tissue was principally restricted to the skin and subcutaneous tissues, not invading the muscles. The microscope revealed a cavernous arrangement of the vessels, and an abundant cell-proliferation in and thickening of the connective tissues.

Although I have called this a case of teleangiectasis, I would not wish it supposed that I consider the excessive vascularity as due solely or principally to inordinate dilatation of pre-existing vessels. I have no doubt that there was a considerable evolution of new vascular tissue.

One of the most striking features in the progress of the case was the gradual spread of the vascular disorder over the whole surface from a few specks in the left groin at birth. Its preponderance on the left side, maintained throughout, seems out of harmony with the small size of the vascular trunks. (Whether the rete mirabile replacing the femoral and external iliac vein was an error of development, or whether it was an acquired condition depending on obliteration of existing normal vessels, I shall not discuss.) That such an extreme disparity might conduce to engorgement of the peripheral vessels will be manifest from the consideration that the inordinate development of peripheral vessels would be attended with a corresponding disproportioned increase of their collective sectional area over that of the feeding artery. This,

in turn, would give rise to slowing or weakening of the blood-current, and so favour the accumulation of blood in the capillaries and veinlets, which would be increased by the hindrance to the effluent current offered by the disproportionate narrowness of the common iliac vein. Such sluggishness of current and ponding back of blood would favour its stagnation and clotting, and also promote the retention and accumulation of effete matters in the limb.

In these ways it might occasion plugging of vessels, accompanied by collateral flexions and œdema, and death of the obstructed vascular territory—the sloughs and ulcers probably were due to this—and it might also induce the repeated attacks of erysipelas, each accession occurring when a certain quantity of effete deleterious material had accumulated. The overgrowth of the bones and other connective tissues of the limb is simply an instance on a large scale of the hypertrophy which is daily seen within smaller limits wherever overfeeding is induced by an increased afflux of blood from any cause, as the presence of a sequestrum, &c.

A

CASE OF SCLEREMA ADULTORUM.

BY

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As the following case appears to me both rare and interesting, I am induced to offer it for the consideration of the Society.

E. K—, the subject of the present communication, is now some forty years of age. She is a married woman. Her circumstances are moderately easy, her hygienic conditions good; her habits are wholly domestic. She may be said to belong to the lower trading class.

The patient was first seen by me in the autumn of the year 1875. My attention was then drawn to large dark patches which she had upon the thigh, and also, as I afterwards found, on other parts of the body.

She had then recently returned from the seaside with irritation and œdema, which was attributed to the moisture and inclemency of the coast. The œdema reached half way up the leg, being moderate in amount and pretty

firm ; it was attended with some stiffness and smarting in the feet and hands, spoken of generally as "inflammation."

The patient is of medium stature, of late years inclined to fulness of figure. She is a rather dusky brunette, without admixture, so far as could be ascertained, of foreign blood. Married early in life, she is the mother of several children.

As cause of her complaint she assigns a mental shock received when six months pregnant. This happened about four years ago. Her previous health had been always good ; but since this event she has become a chronic invalid.

The stains or patches were about thirty in number, varying in size from a shilling to three or four inches in diameter. They might have covered a twelfth or perhaps a sixteenth part of the body. Their figure was mostly oblong or rather square ; generally they were dark and hard. A duller shade was found at their circumference, and near and about them the skin appeared to be darker. As to outline the patches were tolerably distinct. Where pigment is naturally abundant, in the nipples, lower belly, &c., all such regions were abnormally stained, but no heaviness of aspect clouded the face, no dark circle surrounded the eyes, which were indeed of a natural liveliness. The expression was cheerful and fresh ; the complexion moderately clear, and the hair, though black was neither dull nor coarse.

As to colour, the patches exhibited a great variety of tint—dark brown, autumn leaf, purple, lilac, red, light brown, yellow, lemon, and flesh colour ; one might even add white, but with much of a yellow tinge. A dark colour generally predominated, but with some intermixture of red. In many there was found a distinction between the circumferential and the enclosed portion ; the latter was sometimes very sensibly depressed, that is to say, to the extent of half an inch and more, but estimated as very much deeper by the patient.

Two principal patches were situated on the outer and upper part of the right thigh. These, indeed, were the first discovered, and were also among the last to disappear. In the larger of the two patches the central portion was more yellow, while the circumference was of the darkest shade of brown and was also exceedingly hard. The enclosed portion was extensive and much depressed. It was more than two inches long by an inch and a half broad, and the depression was certainly greater than half an inch. Nowhere was it more sunken than at the upper part, where it was bounded by an abrupt and overhanging edge of black or dark brown colour. The surface was coarse and glazed like the rind of bacon or a piece of old parchment stretched.

In this patch the outline was unbroken; the one above it displayed at the edge some short radiations, agreeing in this feature with the "keloid" of Addison.

It may be further said of these patches generally that they were the seat of anæsthesia, of hardness, of pruritus, and of a painful tightness or constriction; less generally, and quite unequally, they were accompanied with tenderness and pain.

The anæsthesia, unmistakeable throughout, was more marked, or at least very strongly marked in the early period of the complaint: the patches then felt dead and numbed, and scratching them, even to the point of laceration, produced no notable sensation or protest on the part of the patient. When this condition had subsided and local tenderness or simply a pruritus ensued, the hopes of the patient revived; she looked forward to amendment and cure. It must be said, however, that in the course of the disorder a degree of anæsthesia has been present all along, not only local but also general. Her movements becoming clumsy, she often received a bruise, and when the injured part was painful she derived some hope from this as indicating improved sensation; her natural feeling, she thought, was coming back to her.

In like manner with the anæsthesia, what was felt of

pain came early. In the upper and outer part of the thigh it was mistaken for sciatica and accompanied with the troublesome numbness, it hindered motion and progression. It was over the painful spots that the dark patches speedily appeared. The development of other patches, it would seem, was not attended with the same kind or the same amount of pain.

In the course of the complaint some such suffering as attends on gout and rheumatism was never entirely absent, due in great part, no doubt, to tension of tissue. With the exception of a dragging on the hamstring tendons this was not intolerable. On the other hand, as the disorder proceeded certain indefinite pains arose. They were described as resembling the effects of searing with a hot iron and as a painful trickling in the flesh; besides these, dartings and stabbing pains, occasionally transient and of short duration, at other times more constant and obscure, were experienced. They seemed to anticipate the sclerosis, with its other accompanying features, as impaired sensibility, &c.; I remarked this particularly in the chest.

A very principal feature in the case is the incessant pruritus; it has attended the complaint throughout, and has never been absent for longer than two days, but raging with unequal intensity. It comes on suddenly and intensely, it increases towards bedtime, and in the course of the night it arouses her from the deepest slumber. It is heightened by contact with water and also by cloudy weather.

The hardness in the patches was considerable; they were described as being "hard as a board." When subjected to friction by the hand there was no sensation in the part, and a subcutaneous core-like thickening might be felt as rolling under the palm.

The constriction was not much noticed at first, though a tightness over the stomach was complained of, and a certain amount of stiffness in the extremities; the constriction, indeed, was more developed at a subsequent stage of the complaint.

As to locality, these stains were more on the anterior than on the posterior aspect of the body, more on the lower than on the upper limbs, pretty equally distributed on either side, but slightly more numerous on the right. On the upper regions of the thighs they were numerous and extensive, and it might be suggested they lay pretty nearly on a level with the genital organs, which are subject, as is well known, to accumulation and also to displacement of pigment during the process of child-bearing.

A patch may be noticed on one breast which was not seen till a week after confinement, when it was found to be quite hard and black. There are two patches just beneath the breasts, two in the front of the neck, one at the angle of the right scapula, all secondary in point of size. There are two large patches at the bend of the waist on either side, the borders being furnished with apparent thongs or tongue-like projections distinct enough in colour, but not sensibly raised above the level of the skin. These prolongations would appear to be concerned in the painful constriction of the corresponding parts. There are two other patches that lie a little lower than the waist. There are large patches upon the thighs, on the outside and inside alike; there is one over either tibia large and symmetrical, extending lengthwise on the whole front of the leg, in colour, so far as the interior space is concerned, of a yellowish white, resembling the bone handle of a worn-out table knife, the frame or surrounding portion being of a delicate flesh colour. On the left leg there is a large spot with something of a vermilion hue. On the left arm and right knee are small patches which in their totality are depressed, admitting the end of a finger or thumb. The others call for no remark. In none of these was there scurf or exfoliation of any kind, nor did they lead to the formation of a scar. The surface, if glazed, was always dry, though elsewhere perspiration was remarkably free. Sebaceous matter was nowhere highly abundant.

I must not omit to mention the œdema which appeared

at this particular phase of the malady. In the summer of the year 1875 the patient went to Great Yarmouth for improvement of her health. She found the weather and climate alike uncongenial, and experienced nothing but discomfort at the seaside. She says that this visit "brought out" the complaint. With œdema of the leg I discovered a pasty feeling present in the trunk, which also pitted when pressed with the finger. The indentation was remarkably white and slow to resume its colour; an elastic worn round the arm betrayed the same condition existing there, and soon there appeared for a time a puffiness in the hand. These symptoms contrast strongly with subsequent conditions. By the next spring all œdema had disappeared, but so long as it remained it was accompanied with tenderness: she was propped at this time with pillows on her chair. Even up to this date she shared in the household management, but in peeling a potato she could not feel it as held between her fingers; she could not sew or pick up a pin. It may be observed that the patient had naturally an arm of comely shape, full and round above and tapering below; both arms kept their colour pretty well, but now there commenced in these parts certain linear irregularities and nodular projections, with subcutaneous thickenings, especially in the left limb.

Here, indeed, is the place to speak of a great change which with advance of the disorder and synchronously with the disappearance of the œdema took place during the winter months and during the spring of the year 1876; that is to say, with disappearance of the œdema and of much subcutaneous tissue, the hardness and discoloration of the patches became diffused, while the depressed portions of the skin were raised. The last to remain defined was the large patch on the left side of the waist. The change occurred with loss of adipose and other tissue to the amount of not less than two stone, a result to which the medicines taken may in minor degree have contributed. In fine, the patches, no longer localised, communicated their qualities to the entire surface; they disappeared insensibly though

unequally, and the whole skin became in a manner sclerosed; the hardness mounted towards the chest and neck, and only the head escaped. The symptoms hitherto displayed, whether we speak of them as morphœa or as keloid, gave place to a diffused and general sclerema—the so-called sclerema of Thirial.

The patient became very dark, especially below the waist and in the extremities. In depth of colour the skin resembled that of a negress, or one of the darkest races: but the staining was otherwise than uniform: in the darkest parts there were yet darker portions interposed. Thus, in spite of glossiness of surface the aspect was singularly disagreeable, and the well-washed integument was always spoken of by her as being dirty.

The locality of the joints was early indicated by an increased depth of tint; the neighbourhood of the coxal articulation was shown externally by a huge black stain. In the course of the limbs there were superadditions to and renewals of colour in the form of markings and blots; there were smudges and alternations in the tint as of brown and yellow-ochre side by side, crowded together, and but slightly blended. The skin is thus varied and unequal, and though the *ensemble* is very black, it is broken with dingy shades of red, purple, or green.

The intensity of the disease having passed to the extremities, the constriction at these parts was very notable. They acquired an unyielding and contracted appearance, being at the same time dark and attenuated. The legs in losing their shape became literally hard as boards, and as to colour were often compared with mahogany. The arms, too, were crippled and drawn up, according to the expression of the patient they “felt as if carried in a sling.” She cannot straighten her legs in bed, and feels them jerked and pulled on by the hamstring muscles with exceeding discomfort and pain. When she wakes in the morning they feel “just like logs of wood.” If her hand is not covered by the bed-clothes “it sets cold, stiff, and numbed, just like the hand of a corpse.” This condition extends up

the limb, and is only recovered from late in the day. In the fingers there is neither feeling nor use, there is pain in the muscles and bones, the tendons are shortened, the aponeuroses apparently contracted, the belly of the muscles is warped and hard, their edges are felt tense and strained; the joints are painful and limited in their play; they are hard but rather broad and square, owing to the tension. The pulse at the wrist cannot be reached, the joint itself is stiff and straight, pronation is impossible. The fingers are fixed in a semiflexed position. Where strained by bony projections and apophyses the skin over the joints, which is otherwise exceedingly dark, displays the clearest white and seems as if soon it would burst. With this condition the soles of the feet and palms of the hands are altogether of a lighter shade, precisely as obtains with the negro race.

It need scarce be said that to pinch the skin or to raise it in a fold is impossible: the natural lines of the integument are lost.

In the upper part of the limbs the constriction is not so tense, and still more does this hold good of the trunk, which on palpation appears pretty soft; the patient still preserves some embonpoint, the colour also is here less dark, being mostly bronze or tawny, especially in the upper part of the body; both these qualities, however, are modified with advance of the disease; the colour seems generally proportionate to the constriction, and as if dependent upon it, nor do I find them dissociated. A sensation of tightness affects the abdomen, and also the thighs and hips, but nowhere causing such distress as is experienced over the stomach and around the waist—a condition not easy to be explained. “She feels,” she says at times, “as if she should burst.” Such discomfort is only transient. When the sclerema had ascended to the neck this feeling was superseded, as it would appear, by recurrent attacks of dyspnœa.

One may remark a mobile or shifting character in this complaint, an alternation and inequality in its incidence,

particularly as regards the limbs. Thus, the left arm began to be affected before the right, the legs are of unequal length owing to the inequality of the contraction. When the arms were most affected the legs were somewhat improved. Thus, though all parts seem to suffer, it is not simultaneously, or with a like amount of intensity. One may say, however, that the right side is still somewhat the worse, and it was also the first affected. As yet the head seems to be spared. This changeful character in the complaint is much remarked upon by the patient. It is well seen in the dark patch upon the thigh; the central portion, which was light in colour and also much depressed, is now on a level with the surface, and has become of the very darkest shade, appearing as a huge black blot. On the other hand, the black rim that encircled it, which for hardness might have been compared to a collar of brawn, is now a flat pale ring about half an inch in breadth, level with the surface, and of a light yellow hue. The distribution of colour is precisely reversed, and the same is true of the seat of the induration.

From observations I was enabled to make at this period of the complaint, and notably in the spring and summer of the year 1876, I became aware of its tubercular character, for which I was not at all prepared. My observations were chiefly made on the left arm, as at that time most open to inspection, and what I perceived was this:—Under influences not well ascertained, but of which weather was evidently the chief, there arose certain tubercles, some of them deep in the tissues, some immediately under the skin, some in the substance of the derm, being sessile and superficial.

They are called variously “knots,” “kernels,” and “lumps” by the patient; they are not confined to the limbs, and they imply activity and advance of the disorder. As observed by me they approached the surface from the deeper parts, and pushed the skin a little before them; they came with throbbing and pain; very commonly they were of the size of a nut, and the skin over

them was black and stretched; the centres sometimes exhibiting an absence of pigment on the surface. They throw out lengthwise on a limb, or they ultimately produce a corresponding black smudgy discoloration which persists as a stain, more deep in hue perhaps as it contracts in size. In a word, the tubercles as they subside are commonly followed by a stain. On the invasion of these tubercles the darkness of the region is intensified, as is well seen in the dark colour round the joints, which on their approach assumes a deeper shade. In this way, or partly thus, as it appears to me, the dark colour has become diffused.

There was a regular succession of these tubercles. They came in every part, impeding motion and causing unrest; they appeared between the breasts, and the stays could not be worn; on the ribs, and they had to be fenced off with cotton wool; they came in the palm of the hand, in the ball of the thumb, the sole of the foot, or its edge, more perhaps on the flexor than on the extensor and supinator surfaces of the limbs. They may affect a linear disposition, not distinguished from the surrounding colour of the skin; of the size of a pea, they will counterfeit a syphilitic form, not circular in their arrangement, however, but straight; when subcutaneous they resemble very exactly a thickened absorbent or vein. Frequent and multiple on the limbs, upon the body they are larger and appear more rare, perhaps because less observable. The patient has affirmed that with the flat of the hand applied, as she moves in bed she feels them roll under the skin.

I am unable to assign a duration to these tubercles. I have seen some of them last a month, and others certainly for a much longer time. A few eminences like tonquin beans appear as sessile growths; they come, and fade away very slowly. Somewhat later I have seen ridges that broke up into tubercles longitudinally and diagonally disposed, and after this, superficial striæ formed, multitudinous, and arranged in every possible direction in the arms, legs, and inside of the thighs. The integrity of

the skin was subsequently ill maintained. There ensued a sort of sudamina or eczematous exudation, with an occasional bulla, pouring out an ill-smelling fluid; a condition which only lasted ten days, and which has only been feebly recurrent. In this I hoped to find a turning point in the disorder, and the tension was, indeed, temporarily relieved. Here and there ensued a nacreous scar of the size of a walnut, of contracted and irregular shape; the limbs have since settled down into a fixed, crippled, and attenuated form.

Somewhat later at the close of the year I had to notice the occurrence of leucasmic and carbonaceous puncta, especially on the thighs and trunk, and of an irregular shagreen appearance in some portions of the skin, the summits of the small eminences being tipped with a pearly white. Large dark patches somewhat resembled gangrene with this leucasmic intermixture; finally, I had to observe, chiefly in the limbs, a condition of the epidermis, which simulated dirty limewash or whitewash on a wall; when scratched with the nail there was made a clear white chalk-like line or tracing upon a dark ground. This is seen alike in the darker and lighter portions of the limb, but it is not possible to remove this surface by washing, and the scraping of the dark portions would only serve to make a wound, the epiderm, though thus dry, being normally adherent. The result is even a dirtier appearance than before. The neck is now sclerosed and discolored within the limit of a dark red line on the level of the inferior maxilla. Some large tubercles are felt in front of the chest. The upper part of the body, the face excepted, is of deep yellow tinge, and free from any appearance of desquamation, the hands are generally free or freed from scale, and have a rosy transparent appearance.

The complaint is alleviated by warmth and also by food; it is aggravated by abstinence and cold. The patient is very much better after a full meal. With returning perspiration in a part she feels assured of its

amendment. The face and head remaining free from sclerema, the perspiration of this region is excessive, large drops rolling off the face at night and soaking the pillows with wet. Under agitation and exertion this secretion is much increased. The feet and hands at the present date perspire freely, as well as the back and axillæ; and it appears as if these parts did the whole of the perspiratory work.

The circulation of the patient has seemed from the first rather wanting in force; the pulse at the temple is exceedingly feeble.¹ The functions of organic life appear otherwise to be little affected. Fever and anorexia are exceedingly rare, and of quite exceptional occurrence. She sleeps moderately well. The tongue has always been clean and unrelaxed, but generally pale. The bodily temperature in a warm room and with a perspiring skin is moderately high. In the armpit 37.5° Cent.; in the feet 29.5° Cent.; in the hands 33° Cent., with one degree of difference between the feet and knees. The muscular irritability as tested by Faradaic electricity was remarkably defective, requiring a very strong current to produce recognisable contractions of the muscle; the effect did not exactly correspond to expectation or to existing intensity of the sclerema.²

¹ In a sitting posture, as ascertained by means of the stethoscope, the pulsations have been 90 to 95 or more; the excitable character of the patient has served to modify this computation.

² I was happy to avail myself of the experience of Mr. William Sedgwick, in the application of the Faradaic current, the more as he had seen the patient with me previously, and his well-known published case of keloid gave him an interest in the subject. It was found that muscular irritability in the right arm was greatly reduced, and still more so in the left. It was very little to be detected in the right leg, while in the left leg it was entirely abolished. These results were very decided. I was deterred from repeating the application by the pains which followed it. These continued for some days, and were of the same nature as those already instanced, viz. burning with a hot iron and recurrent stabbing pains in various parts of the body and limbs, more, perhaps, in the legs than in the arms. I am also indebted to Mr. William Sedgwick for the recognition that these pains were not dissimilar to those that are found in progressive motor ataxy. The precise relation of these

The action of the bowels was moderate ; of late more liable to be acted on by diluents as beef tea, and especially by the compound decoction of sarsaparilla which purged her violently. The urine was sometimes scanty, generally high coloured and thick with lithates, and not unfrequently dark like strong tea or rather a thin weak coffee. The catamenia were scanty and pale, more than once like dirty water. The constitutional habit is bilious and nervous ; the family history is disadvantageous, on the father's side bronchitis and consumption, on the mother's asthma and dropsy. There was no history of rheumatism and gout : before marriage she worked in a very hot laundry, and conjectures it may have impaired her constitution.¹

The complaint is unhesitatingly attributed to a fright on hearing that one of her children had fallen out of the window. She was supposed to have hurt herself in swooning, and got about painfully and clingingly up to the date of her confinement. In parturition she could not shift herself and had to be lifted from side to side ; the accoucheur inquiring about paralysis in her family. In the act of parturition the brown patches were discovered and were then mistaken for bruises. She rubbed the places with warm oils. She suckled the child a year, but could not carry it, nor could she move along the street. Finally, being threatened with dropsy (œdema), her mother's complaint, she was induced to apply to me.

A very remarkable feature is the extreme sensibility to cold, and more especially to damp or contact with anything wet. She anticipates by her suffering every turn of the weather in that direction. In no other patient have I observed an equal sensitiveness. A dirty rainy day

pains to local tenderness, sclerosis, and impaired sensibility in given areas, though made the subject of attentive consideration by us, I deem too uncertain and too little confirmed to be here insisted on.

¹ As to complexion, her father as well as her husband were very dark. Not all of her mother's family are dark. Her eight children, all girls, are fresh looking, and inclining but slightly to a dark shade. Two have rachitis and one torticollis. The patient has two black moles on the bosom, about the size of a pea, but the daughters are not remarkable for moles.

is most trying. She is then quite beside herself with pruritus and feels "oh ! so stiff in joint and limb." At such times the stiffness affects the face, and it has seemed to spread under impulsion of this cause. A dry March wind afflicts her less than rain and damp in summer ; but sitting in a draught or a moderate degree of cold will make her powerless. No decline, however or retrocession of her complaint has been remarked in the summer season. The use of steam vapour has been attended with some present relief, but after having been long employed for the arms, it had to be abandoned ; they were found to be stiffer afterwards and were at times afflicted with sudden and violent twitchings. The contact of water has always been followed by distress. The daily ablutions were a source of extreme discomfort ; causing stiffness and an icy coldness, after which come smarting and a most terrible and continuing pruritus ; at such time the limb will sometimes take a shade of green. At the seaside she could not walk on the wet sand ; the spray of the sea was quite a terror to her as the source of intolerable suffering, and the smarting produced was past description.

A remark has been made by those about her, which is also in accordance with my experience—that the colour varies in intensity from interval to interval of visit, and even from day to day. There is no pungency of smell about the patient, but a sickly odour is discoverable like that of an unwashed baby.

I must not omit to instance as a prominent feature of this complaint, the existence of those crackling sounds in the joints which are held to be characteristic of rheumatic gout.

Of remedies employed I found that tonics generally disagreed ; this was peculiarly the case with iron, which caused headache and increased sclerema ; arsenic could not be long taken ; and quinine generally disagreed. The only medicines from which she has experienced decided relief are those which act on the liver and kidneys, principally Bayley's pill (Pil. Hydr., Pulv. Scill. et Digit.) and colchicum ;

the former I have given rather freely ; but my present inclination is to dispense with medicine as much as possible. The patient has a liberal supply of food and at least half a bottle of thin claret daily ; for dyspnœa she is supplied with æther and is allowed a trifling portion of spirits.

APPENDIX.

In order to continue the history of this interesting case as far as possible in this communication, I have thought it desirable to add the following :—The condition of the patient as to physical power has been feebler than at the beginning of the year, and occasionally her sleep has suffered and her appetite has seemed altogether to fail. She has now kept her bedroom seven months. During that period there has been seen an increase of the cuticular condition already spoken of as resembling dirty limewash and more especially of late a very great increase of the dyspnœa which from its intermittence would seem rather of a spasmodic character than dependent on any internal organic change ; it is not accounted for by any physical signs.

The hands remain fresh-looking, rosy, and pink. The colour of the lower part of the body is slightly improved, being mostly of a deep brownish-yellow shade. Of open lesion there is nothing more than a low inflammatory desquamation at the root of the thumb, and over one of the ankles a honeycombed abrasion or trifling wound.

The temperature at present (July) is fairly high, but without pyrexia, and accompanied with a fair amount of perspiration, increased in the armpit, feet, and elsewhere, but considerably diminished in the face. I measured recently in the mouth 37° Centigrade, armpit 37° , palm 34° , feet 30° . The pulse ranges from 100° to 104° , being somewhat higher than at an earlier date. It is true she has passed through a season of anxiety, principally from recurrence of dyspnœa,

which occasioned very great distress by frequent paroxysms, and with it there was sleeplessness, failing strength, and anorexia. These paroxysms ceased suddenly as if with change of weather and have rarely recurred. The sclerema has now extended to the face and is constantly present, but gives no inconvenience; in advance of the masseter on either side there is seen a marked depression, not permanent, but occurring alternately first on one side and then on the other. The tubercles are no longer produced on the extremities, but there remain two large subcutaneous knots anteriorly in the region of the waist. The legs and lower part of the body are still very dark, the neck is brawny and firm. Over the back and shoulders there is an extensive leucasmic interlacement which diversifies the yellow or bronze colour; this denotes here, as elsewhere, an advanced stage of the complaint. The condition of epidermis hitherto spoken of as limewash or dirty white-wash has much augmented on the limbs, and especially on the forearms and legs. As a last word, we may speak of it as tending to decline.

The legs and forearms have undergone almost complete muscular atrophy, and present a withered aspect when contrasted with the thighs and upper arms, as the following measurement will show. Around the middle of the thigh $16\frac{1}{2}$ inches, calf $10\frac{3}{4}$ in., instep $9\frac{1}{2}$ in., ankle $7\frac{1}{2}$ in. Round the arm 12 in., forearm $8\frac{1}{2}$ in., wrist $6\frac{1}{8}$ in. The fingers and wrists are completely fixed, and the movements of pronation and supination are no longer possible. The right elbow is as if ankylosed at a right angle, but still admits of a very slight amount of flexion. The left joint is capable of a little more movement. The knuckles are extremely curved, tense, and to all appearance irrecoverably fixed. The fingers are shining and somewhat rosy, and except as above stated show no tendency to desquamation. The nails continue strong, the hair shows no weakness, the eyebrows exhibit no change.

Besides the large nacreous cicatrices already mentioned as resulting from exudation of a prior date, there are now

seen others of a more superficial character, in size and figure not unlike vaccine marks when these are not deep, and have besides a punctated surface. These also denote the seat of previous exudation. Such scars are very noticeable on the fleshy part of the arm below the shoulder on the right side. They are here found in a group, but are not so obvious elsewhere.

One cannot but remark that both in the dyspnœa and in the limewash character of the epidermis there is an approach to leprosy, which is also exhibited in some other symptoms, as the extreme sensitiveness to weather, the leucasmus, &c. The sclerema is so far general that only the scalp and forehead have escaped. It is to be observed, however, that as yet there is no increase of pigment in the face, the appearance of which is almost normal, nor is the induration here of such amount as to interfere with labial pronunciation. The buccal cavity and vulva seem in no degree affected; the axilla and groin are softer than the surrounding parts, but have participated in the sclerema. The disease has spread from separate centres, and its early limitation by defining lines was conspicuous enough. There is at the present time a lull in the more prominent symptoms and a tendency to restoration of the normal colour, and this is especially seen in the arms; and these facts, taken with the absence of increased pigment above and a minor degree of induration in the face, afford a hope that the disease has met with an arrest. As yet I know of no instance where the whole surface has been affected as is threatened in the present case.

A CASE
OF
SIMPLE ATROPHIC SCLEREMA,
ASSOCIATED WITH
DISORDER OF THE CIRCULATORY AND ALIMENTARY
FUNCTIONS.

BY
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(Received December 11th, 1876—Read January 23rd, 1877).

THE following history will form a fitting supplement to the preceding paper by Mr. Gaskoin, as, while the case presents many points of agreement, the apparent differences are probably only those which may be expected to result from a difference in the exciting cause, and to the circumstance that in Mr. Gaskoin's case the morbid action has hitherto chiefly affected the peripheral parts, while in my own case the central organs have been from the first more deeply implicated.¹

William W—, æt. 45, a stonemason, came to me in March, 1872, complaining that he was no longer able to work from inability to close the hands upon his tools.

He stated that the disease began with "a little cold and

¹ I am indebted to Mr. Gaskoin for opportunities of studying his most interesting and instructive case.

indigestion," in the preceding September, when he was taken with a sensation of "great weight" in the epigastrium after food. It was accompanied by "terrible retching," but nothing was ejected except a little watery fluid. Under medical treatment the symptoms subsided, and then the hands gradually assumed their present condition.

He has remained constantly under my care up to the present time, so that my observation of his condition has extended uninterruptedly over a period of more than five years.

Speaking generally, the hands have undergone no change, but his health has declined, and changes associated with those affecting the hands have been gradually developed during this period.

The patient, now in his fiftieth year, is of moderate build. He is fair and has blue eyes, scanty reddish whiskers, a bushy head of hair just turning grey, and a thin, delicate, and only slightly pubescent skin; the lobes of the ears are prolonged and adnate.

Although he has latterly become somewhat anæmic, he still retains traces of a naturally ruddy complexion, due to the permanent injection of fine networks of minute cutaneous vessels.

He is the father of five healthy children, aged twenty-three, twenty, seventeen, thirteen, and nine, respectively; he has lived a careful, temperate, and industrious life, and has suffered but one illness, a severe attack of well-ascertained typhus, ten years before the present condition came on. He was able to resume work about thirteen weeks after he succumbed to this fever. His recovery was complete and was retarded by weakness only.

From the age of twenty-five to thirty-seven, he was occasionally troubled with small sluggish boils appearing about the back of the neck, and the white scars of two or three of these boils are still present below the nape.

His normal weight is about eleven stone, with very slight variation. He considers himself to have possessed average muscular strength, but from boyhood he has had

a tendency to faintness with outbursts of perspiration when in a close atmosphere. He has also been liable to flushings, and has always perspired freely, and on the least over-exertion, profusely. He has smoked since the age of twenty, his usual daily allowance of tobacco being from a quarter to half an ounce. He never experienced uncomfortable effects from its use, and he still smokes about half of the above-mentioned quantity.

His remote ancestors were mostly long-lived ; his father and mother died early, but all their children are alive and moderately strong.

The father died after eight months' illness, at the age of thirty-eight, and when William W— was fourteen years of age, "of enlargement of the heart followed by dropsy." His son does not know whether it was of rheumatic origin or not. The mother experienced an attack of bilious and nervous fever (probably enteric) shortly after her marriage, and was ever afterwards a delicate woman.

She married a second time, and the issue of this marriage, a son, is alive and well. She eventually died of "consumption" at the age of fifty-one.

To return to William W—. When he first came to me, and for a year or more afterwards, he presented nothing unusual in appearance, nor, perhaps, would a casual observer have noticed anything amiss with his hands. There was no enlargement or oedema of any part of them, but on near inspection, the fingers from the first joints to the tips were seen to be sometimes rosy, but more commonly dusky, and on some days livid ; and on taking either hand into your own it felt from the wrist downwards as cold as that of a corpse. The fingers were rounded, smooth, and hard, like tendon ; no pitting could be produced by pressure, and it was impossible to pinch up the least fold of skin, the mobility of which was reduced one half, and it appeared to be tightly drawn over a hard substratum, so that the hands were constantly kept open, and it was impossible to close them completely.

After prolonged efforts he could, indeed, bring the finger-

nails of the left hand in contact with the palm, an inch and a quarter below the upper border of the hand (the anterior fold of the wrist), but he could not bring any nail of the right hand in contact with the palm, and after the strongest effort the finger-ends impended at the distance of an inch over the upper limits of the palm.

This induration of the areolar tissue was marked around the wrists, and gradually ceased about four inches above the joints.

The wrists were kept straight, and were still capable of limited movement; coercive attempts to flex or extend the hands caused pain, and the slighter disturbances incurred by their necessary use produced an intermittent aching, which was much complained of. The rotatory movements of the forearms were reduced to one half, *i.e.* the plane of the palm in passing from extreme supination to extreme pronation, described a quadrant only.

A fine creaking or grating is often felt immediately under the skin, by a finger placed on the back of the wrist when it is in motion.

The hands perspired freely, and the fingers were usually damp—some days even wet—as well as cold.

The actual temperature of the fingers was usually from 3° to 5° only above that of the external air; and from 30° to 40° below that of the axilla. After sitting in a warm room before the fire, and when warm in bed, the temperature of the fingers has become nearly 10° higher than that of the external air.

These facts, and the comparative temperatures of various parts of the body, are given in the accompanying table.

Temperature (Fahr.) of the fingers and hands, as compared with that of the apartment and other parts of the body.

| | Air. | Fingers | Palm. | Axilla. | Mouth. | Toes. | Sole. | Ankle | Remarks; condition of fingers. |
|----------------|------|-------------------|-------|---------|--------|-------|-------|-------|--------------------------------|
| 1872, Sept. 18 | 60° | 60° | 65° | 98° | ... | 81° | 82° | 83° | |
| Oct. 2 | 60 | 65 | 66 | 97·5 | 98 | | | | |
| 10 | 60 | 65 | 70 | 98 | ... | 65 | 75 | 80 | |
| 16 | 59 | 60 | 63 | 93 | ... | ... | ... | ... | Very dusky. |
| 23 | 58 | 63 | 63 | 97·2 | | | | | |
| 30 | 57·2 | 63·5 | 65 | ... | 99 | | | | |
| Nov. 13 | 52 | 55·5 | 59·25 | ... | 100 | ... | ... | ... | Very livid. |
| 1873, April 30 | 54 | 60·7 | | | | | | | |
| 1874, Sept. 22 | 67 | 66 | | | | | | | |
| Oct. 6 | 61 | 64 | ... | ... | 90 | | | | |
| 1876, Aug. 15 | 61·5 | 70·5 ¹ | ... | ... | 99 | | | | |

The sense of touch was very slightly, if at all, diminished; and the tips of the index fingers, which certainly had their full share of the disease, maintained their natural superiority. Thus, the limit of confusion for the whole hand occurred when the points of the compass were less than two lines apart; whereas the tips of the index finger discriminated two points when they were separated by an interval of only a line and a half. The nails were perhaps a little more incurved at the edges than usual, but their nutrition and growth were normal.

Such, I repeat, was the state of the hands nearly five years ago, and as there were no other indications of disease at this time, I was greatly puzzled as to the nature of the local disorder. I was inclined to attribute it to disease of the arteries of the hands, leading to dry gangrene, and thought of the stories of ergotised grain. But the patient had always resided in London, and the radial arteries, in common with those of the rest of the body, were soft and full. Subsequently, I was satisfied that the impediment to the circulation was due to the induration of the integument, which necessarily interfered with the movements of the arteries. From the further course of the disease it

¹ After sitting two hours before a fire.

may be assumed that the adventitia of the arteries, at least, was involved in the sclerema, thus preventing the expansion of the vessels.

Up to the present time the hands have remained unchanged, excepting that a hard, linear scar has formed obliquely across the palm, firmly binding the skin to the subjacent fascia, and that gangrene has actually declared itself, although in the very slightest degree. A year ago the first indication of this change showed itself as a minute, depressed brown scale on the extreme tip of the right middle finger, attended by sharp throbbing and shooting pain in the part. A speck-like cicatricial shrivelling now marks the seat of this change. Three months later on, the tip on the right index finger was similarly affected, and healing was attended by the discharge of a little thin pus. In November last this action was renewed at the tip of the same finger, and was accompanied by a moderate degree of inflammation, resulting in the separation of the cuticle of the forepart of the finger. A little discharge still continues; the forepart of the nail is separated, and the finger tip is slightly truncated.

The face was observed to be gradually losing expression, the mobility of the skin, especially over the malar bones and forehead and of the scalp, decreased, and the subcutaneous tissue grew firmer. Simultaneously the affection spread over the neck and upper part of the chest. The subcutaneous tissue of the abdomen is unaffected, but the aponeurosis of the external oblique muscle at least is implicated, for the moderately protuberant walls of the abdomen are hard and unyielding, and this condition is a source of discomfort to the patient. The fibrous tissue around the knees appears to be similarly affected. The other parts of the body, even the back immediately below the vertebra prominens, are at present free from the disease.

The expression is that of melancholy impassiveness; the mouth is always closed, and the lips somewhat compressed. A permanent hard ridge is seen across the forehead. It is produced by pressure of the hat.

The pleasant emotions may still evoke a ghastly smile, but the opposite passions have no longer power to contract the brow.

The characteristic features are, however, best displayed on the neck. When the head is thrown back, complete extension is prevented by the superficial fascia in front of the neck, which forms a thin, hard, resisting plane beneath the skin, gathered from the sternum and clavicles upwards into slight vertical ridges, feeling like knotted cords. Here the skin is free, but lower down, as far as the second costal cartilage, it is drawn very tightly over the subjacent parts, and in passing from the root of the neck, over the sternal ends of the clavicles and the manubrium, it attains its maximum degree of immobility, and is not only highly polished but partially eburnated in appearance. The surface is still further diversified by pink spots or little patches of minute straggling vessels, rendered conspicuous by the poverty of the skin in which they lie. The sides and back of the neck are brawny. On a cursory examination it appears seamed or furrowed, coarsely about the nape and just above the clavicles, and finely below the ears. The skin, however, is perfectly smooth, and the alteration is due to a partial disappearance of blood-vessels from the naturally rosy surface, which is now broken up, in the regions above stated, into red and white areolæ, varying in size from half a line to three lines in width, and arranged alternately with considerable regularity. The pink areolæ exhibit to the naked eye ramifications of minute blood-vessels, such as are seen in this and other parts of the surface in persons freely exposed to the weather. The extremities of these vessels terminate abruptly in the margins of the colourless areolæ. These latter are of ivory-like whiteness and opacity; here and there a minute blood-vessel from a contiguous vascular areola may be seen streaking it with pink, but usually the colourless areolæ are absolutely bloodless. These characters are clearly and conspicuously displayed under a low magnifying glass.

Towards the shoulders and back the red and white

mottling gradually disappears, the white areolæ becoming larger, more diffused, and, losing their ivory-like appearance, gradually assume that of the healthy skin; correspondingly the pink areolæ becomes smaller and less vascular, until they are reduced to mere points, and ultimately disappear; and thus the morbid and natural tissues are imperceptibly blended.

The skin of the affected parts retains, as far as I can determine the fact, the normal amount of sensibility. A distance of five lines between the points of the compasses is the limit of confusion for the whole of the side of the neck, but when the points are placed on the *areolæ atrophicæ*, at the root of the neck, they are rarely discriminated as two when fully an inch apart.

The skin generally has a slightly freckled and somewhat dirty appearance, particularly about the sides of the chest, due to a little increase of pigment. The adipose tissue has entirely disappeared from the integument of the upper extremities, the neck, chest, and back, the muscles of the arms are much wasted, and the borders of the scapular muscles are apparent under the thin skin. The lower part of the body and the lower limbs are in fair condition, and if the muscles of expression, the platysma myoides, the lumbricales doubtless, and perhaps the external oblique of the abdomen, be excepted, the muscular system must be regarded as free from disease. The wasting of the muscles of the upper extremities is clearly due to inaction.

Thus much respecting the local disease. I pass now to the general condition of the patient. He is weak and anæmic, and the neck and shoulders stoop. The curvature of the neck is mainly, if not altogether, caused by the contraction of the superficial fascia in front of it producing a forward inclination of the head.

But the most serious troubles are those which arise from associated disturbance of the circulatory and digestive organs.

During the first two years of the disease the pulse was

normal, tranquil, regular, of good volume, and moderate force, and ranging between 70 and 80. Shortly afterwards it began to intermit once or twice in the minute. This defect increased rather rapidly, and attained its maximum during the fourth year, when the heart's action exhibited remarkable variations, which have continued ever since. Sometimes the pulse is only 40 to 42, the beats regular and equal; but on listening over the heart 80 to 84 regular, but unequal ventricular contractions, are clearly audible, a feeble one regularly alternating with the stronger, which alone are appreciable at the wrist. But the most remarkable feature is that occasionally the feebler beats are altogether eliminated, the ventricular contractions being reduced to the same number (40 to 42) as the radial pulsations, the action meanwhile being quite equal and regular.

This regular intermission of the pulse and reduction of the cardiac contractions gives place either to the normal state—a regular pulse of 72 to 80, synchronous with the ventricular contractions—or to the usual form of irregular action in which the cardiac contractions being of unequal force the radial pulsations have a corresponding character with frequent intermissions. This latter is the common state of the pulse at the present time.

Associated with the gastric symptoms, which I will now describe, the patient has severe attacks of cardiac pain, with breathlessness, palpitation, and faintness. In the patient's own words, "at one time the heart seems as if it would jump out of his chest, and at another that it is not going at all," and he "thinks that he shall die."

The cardiac irregularity above described, has from the first been attended with dyspeptic symptoms, varying in frequency, but often occurring twice or thrice a week.

For the last two and a half years he has almost always complained of a pain, variously described as "heavy, burning, and gnawing," across the sternum on a line with the nipples. But occasionally pain of a more

severe character attacks the cardiac, epigastric, and left hypochondriac regions. It is usually accompanied by flatulency, and ends in severe retching and vomiting of sour or bitter mucous fluid.

Apart from these attacks the patient considers his digestion good. They are not provoked by errors of diet, for his food is of the simplest kind, and his drink only milk and water and weak tea. He has a constant tendency to diarrhoea and the motions are rarely formed.

To sum up. The patient inherits a rather delicate constitution, and this was probably somewhat enfeebled by a severe attack of typhus fifteen years ago. He is now greatly debilitated. A walk of two miles is as much as he can accomplish at best, but oftentimes a much shorter journey proves too much for him; "he feels faint as if he had no breath at all," an inclination to action of the bowels comes on, and a little watery mucus is discharged with slight prolapsus of the bowel.

Frequently after sitting in the cool waiting-hall an hour or two, he presents himself in the prescribing room with beads of sweat on his brow. The pupils are moderately dilated; the frontal vein is large and lax; it lies in a hard deep channel, and becomes full and prominent on the slightest inclination of the head, and a dilating wave of blood is at once thrown back into the vessel by rendering the tissues between the malar bone and root of the nose tense by a slight depression of the cheek, thus plainly indicating the complete absence of tone in the coats of the vein. A narrow line of dusky congestion occupies the margins of the gums; the inner surfaces of the thighs are pervaded by a visible network of fine blood-vessels, and an irregular festoon of similar vessels radiating upwards is seen over the anterior margins of the ribs and epigastrium.

The larger veins everywhere appear healthy. It is necessary to state that the patient is free from both varix and hæmorrhoids.

Apart from his graver troubles, and as a matter of

common complaint, he speaks of "a heavy sensation across the forehead, and a sharp pain across the chest."

The lungs and kidneys are normal, and the urine is poor, the sp. gr. being usually under 1008.

Treatment.—I have used tonics and stimulants, general and local, including ample trials both of digitalis and belladonna without avail. He has taken iodide of potassium at intervals, and has spent some months in the convalescent institutions of Bognor and Walton-on-Thames without advantage. He has lost strength in spite of all that has been done for him, and I cannot but regard the issue as unfavorable.

Pathology.—I have described with some minuteness the condition of the skin of the neck, for here, by the help of a pocket-lens, we may read the morbid anatomy of the disease. We see areas, so regularly disposed as to resemble broken lineæ atrophicæ, from which the blood-vessels have more or less completely disappeared, and the soft, clear, pink skin is converted into a firm, opaque, white, and bloodless structure. While the blood-vessels have collapsed and disappeared the system of lacunæ and canaliculi pervading the healthy connective tissue has shrunk; the adipose tissue for the most part has disappeared, and the soft, moist, and open cushion has collapsed into a hard, close, comparatively dry, and ill-nourished tendinous structure.

I hoped to have secured the attendance here to-night of a spare woman, otherwise healthy, in whom the subcutaneous tissue of the inner surfaces of the fingers and of the palms has become everywhere indurated and compacted with the palmar fascia contracting and fixing the fingers and seaming the narrowed and hardened palms. But such cases are familiar to all of us, and the subject of this paper himself exhibits this condition as it is usually seen, the centre of the right palm being seamed, tucked in, and firmly fixed to the fascia beneath.

The nature of the disease in this patient is illustrated very simply and clearly by these common instances of

partial sclerema of the palm, and the close associations which I here recognise will be a great aid in the proper classification of such examples of sclerema as that under consideration, for it may at once be isolated and distinguished from the greater number of the forty or fifty cases which have been described under the term sclerema and its synonyms. Only three or four of these have furnished opportunities for post-mortem examination.

In Förster's¹ case, which Dr. Fagge regards as an example of Addison's keloid, the corium and connective tissue were confused, forming a homogeneous white tissue, from which the fat had vanished, and the sclerosed connective tissue was firmly bound to the muscles, fasciæ, and tendons, and the author regards the change as due to "a chronic process of proliferation in the connective tissue of the corium, and particularly of the subcutaneous tissue, unattended by fever or local inflammatory symptoms."¹ It is implied that there was an increase of connective tissue in this case. Kohler,¹ Auspitz,¹ Arning,² and Day,² expressly state that such was the case in their patients, and they especially mention an increase in the elastic element.

I cannot introduce a comparison with Rasmussen's³ case, for it appears from the progress of the disease, as well as from its anatomical characters (free cell infiltration of the connective tissue), to have been a case of cancerous induration of the skin.

The question of hypertrophy of the skin and subcutaneous tissue, when these have coalesced, excluding due consideration of the adipose element, is one that must be answered very cautiously in any case, for it is certain that before we can know that any absolute increase has taken place we must previously know the

¹ Referred to in Day's paper, 'American Journal of Med. Sciences,' 1870, vol. lix, p. 357.

² For references, see Classification, p. 149-150.

³ 'Scleroderma, and its relation to Elephantiasis.' Translation by Dr. W. Moore, Edinburgh, 1867.

thickness and consistency of the pre-existing adipose layer. Of the "proliferation of the connective tissue," so freely spoken of by Förster, neither he nor any other observer gives us any proof. Kaposi, indeed, found in one of the cases under his observation a "stasis of lymph-cells in the perivascular spaces," and he assumes that "this stasis and stagnation of lymph in the interstices of the tissues occurs in the production of scleroderma, the connective tissue being formed in excess out of the accumulated superfluity of nutritive material."¹

That the integument may be thickened and hardened by lymph stasis is obvious enough, but that growth of the connective tissue takes place as a consequence is a pure assumption. Nor, indeed, can Kaposi's case be regarded as typical, for the patient had lupus erythematosus, oedema of the legs, repeated attacks of "urticaria" in the affected parts, recurring rheumatic pains, and thickening and hardening of the bones of the legs—symptoms which are commonly associated with a specific irritation of the lymphatics.

Watching, as I have done, the gradual development of the disease in my patient, I can affirm that there has been no "proliferation of connective tissue" in his case. At no time has there been the slightest enlargement of the affected skin; on the contrary, the parts affected,—excepting perhaps the fingers, which have continued throughout in a chronic state of venous congestion from mechanical impediment to the return of blood,—have shrunken.

The atrophy is, of course, due in part to the more or less complete absorption of fat, but an examination of the skin in front of the neck of this patient, in which the disease is still progressing, will, I think, be sufficient evidence of that for which I contending, namely, that the disease in this particular case is due not to an increase of the subcutaneous connective tissue, but to its atrophy *ab initio*.

¹ Hebra, 'On Diseases of the Skin,' Syd. Soc., vol. iii, p. 123.

But what, we may ask, is the cause of this atrophy of the nutrient canals? of this conversion of skin into tendon? "Doubtless, some affection of the vaso-motor nerves" is the reasonable answer.

That the sympathetic nervous system is generally involved in this case, appears to me certain. The functional derangements of the heart, stomach, and intestines, the lax condition of the coats of the blood-vessels, the easy provocation of sweat, and progressive inanition, are to me marks of a grave depression of the sympathetic—of a slowly advancing paralysis of this system of nerves, of which the subcutaneous atrophy is but the superficial indication.

The case narrated is remarkable for the slow progress of the sclerema, and for its unmistakable association from the first with derangement of the central organs. In the cases recorded, disorder of the stomach or heart, or of both, is mentioned in only four or five.

In Rilliet's¹ case, the disease was ushered in by a sudden and violent pain in the epigastrium, accompanied by very intense palpitation, but these symptoms, as well, indeed, as the sclerema, may properly be referred to inflammatory irritation of the serous surfaces (in which I include the areolæ of the connective tissue), for at the end of a week or ten days, when the induration was subsiding, slight ascitis, and effusion into the right pleura and pericardium were noticed.

In Barton's² case the sclerema was preceded by dyspeptic symptoms, failure of appetite, and sickness after meals, with occasional vomiting.

But the cases which in several particulars most closely resemble that under present consideration, are those related by Dr. Arnold of Baltimore,³ and Dr. Day of New York.⁴ In both of these, derangement of the digestive and circu-

¹ 'Med.-Chir. Rev.,' 1848, p. 79.

² 'Dublin Quart. Journ. Med. Sci.,' Aug., 1869.

³ 'American Journ. Med. Science,' July, 1869, vol. lviii, p. 89.

⁴ *Ib.*, April, 1870, vol. lix, p. 350.

lating organs, were prominent symptoms. The troubles in the latter case were proved, however, both by the post-mortem examination, and by the physical signs during the progress of the disease (pain and fine crepitant râles in the upper part of the lung, and thence to the præcordia, &c.), to be due to chronic pleuritis, pericarditis, and peritonitis.

In the case before us, the disease has entered its sixth year, but there are no positive indications of organic disease in any of the viscera; the chest movements are free, the percussion note is good, and the breath-sounds for one in his debilitated condition, may be considered normal. Expiration is, indeed, slightly audible in all parts of the lungs, and the gentle vesicular murmur of health is somewhat encroached upon by a proportionate increase in the bronchial sounds—changes which, in an ordinary case, one should unhesitatingly refer to diminished elasticity of the lung, but which in this may be regarded as an indication of fibrous degeneration of the lung tissue.

At present the heart cannot be charged with organic disease, and the gastric symptoms are referable rather to defective or vitiated secretions than to peristaltic action hindered by adhesions.

Dr. Arnold's is really the only case which resembles that under consideration, for the two agree both in the duration of the disease and the attendant troubles of the alimentary canal. That these were merely functional in Dr. Arnold's case, appears from his statement, that "the cardiac and thoracic functions were normal, but the pulse was weak and slow."

So far then this case is almost unique, and as the disease is still in its mid-career, further opportunities will be afforded of watching its progress, and of testing the accuracy of the conclusion which I have drawn, respecting its nature and associations.

The slow progress of the disease suggests one other consideration. In most, if not all, of the recorded cases, the peripheral disease—the scleroderma—has been the prominent affection, and few fatal issues are recorded; in

the case under present consideration, the sclerema has become quite secondary to the grave disturbances of the circulatory and nutritive functions. How far the central disturbances interfere with the peripheral affection, or whether they do so at all, is a question worthy of consideration. I find it noted in Dr. Day's case, that an attack of vomiting and purging, lasting some days, was followed by "a great amelioration in the condition of the skin, which lost much of its bronze colour, became softer and more supple, and in parts could be moved over the subjacent tissues."¹ If we may recognise cause and effect here, then we have an explanation why the sclerema in my patient has during these five years made so slow a progress, for vomiting and occasional diarrhoea have never been absent from the first, and during the last three years they have been frequent and severe.

In conclusion, I venture to make a few remarks on the classification of the variety of disorders of the skin and subcutaneous tissue, which have been or may appropriately be included under the term sclerema and its synonyms.

Dr. Hilton Fagge² has done good service by bringing together "Keloid, Scleriosis, Morphœa, and some allied affections," and pointing out the relations between them; but the case which I have narrated to the Society, and that very interesting one recently brought under its notice by Mr. Gaskoin, throws additional light on the subject, and call for some further generalizations.

The terms *sclerema* and *scleriosis* are well suited for embracing these affections; *scleroderma* as a generic term is insufficient, since it implies an affection of the skin alone, whereas the morbid process in every case, involves the subcutaneous connective tissue, and in most, if not all, has its origin in this structure, the derm being usually involved in the later stages of the disease only.

Dr. Kaposi³ defines sclerema (scleroderma) as "diffuse

¹ Loc. cit.; see also the Appendix to this communication, p. 151.

² 'Guy's Hospital Reports,' 3rd ser., vol. xiii, p. 255.

³ Hebra, 'On Diseases of the Skin,' New Syd. Soc., vol. iii.

hypertrophy of the connective tissue," and of course places it in a class quite distinct from "xeroderma¹ or parchment skin," since he defines this as "diffused idiopathic *atrophy* of the skin." He says, "the course and development of sclerema and xeroderma, seem to distinguish them in a marked degree from one another. In the former, the disease commences as a board-like infiltration of the subcutaneous connective tissue, the corium being involved later and in certain parts only, the epidermis not at all, or only in the later periods of the disease. In xeroderma, the shrinking, the atrophy, appear to begin in the papillary layer and the epidermis, and from thence only to spread to the corium. This is evidenced by wrinkling of the epidermis, disturbance of the pigment, and the quantity of small superficial teleangiectases." (p. 256).

He disregards the fact that atrophy of the papillary layer is attended by a proportionate loss of sensation. Thus, in the first case, a girl of eighteen, the xeroderma had existed "from early childhood," but "the sensibility of the skin was not diminished." In the other cases, the disease had existed in one, a girl of ten years, "from earliest childhood," and in the two others, "from the age of twelve months." The tactile condition of the skin is mentioned in the first case only, and it is obvious that if the papillary layer of the derm had been the seat of diffuse atrophy for a period of ten years only, the sensibility of the skin would not have remained intact as it is stated to have been. Hence it may be concluded that the special seat of the disease in so-called xeroderma is not the papillary layer. Then as to the condition of the epidermis, "disturbance of the pigment," "checkered pigmentation," certainly does not exist less frequently in sclerema than it does in so-called xeroderma. In Mr. Gaskoin's case, where xeroderma (ξηρόδς, parched) and sclerema are most

¹ Those authors to whom these high-sounding but trivial words are apparently of so much importance, should be careful to give them their proper meaning. "Xeroderma" no more denotes "parchment" skin than does the term "cutis anserina" or "pachyderma."

completely associated, there is not only confusion, but frequent interchange of all their supposed distinctive characters. As to wrinkling and shrivelling of the epidermis, this is merely the effect of the atrophy of the subjacent corium, and may be taken generally as a mark of the age of the disease. In only one or two of the recorded cases of sclerema had the disease existed so long as five years, and in none of Kaposi's cases of xeroderma had it existed for less than this term, and he had no opportunity of seeing either of them, until the disease had attained this and a greater age; he has, therefore, no ground for assuming that the morbid changes commenced in the papillary layer. Indeed, if supposition is to take the place of fact, it were more reasonable to assume that these cases of xeroderma originated in "Sclerema neonatorum," a condition indistinguishable from the "Sclerema (Scleroderma) adultorum."¹

There is only one more of Dr. Kaposi's distinctions that requires notice. It is the presence of small superficial teleangiectases. These are not infrequent in sclerema; they are characteristic of one form at least of keloid, and they form a very prominent feature in the case which I described.

If the preceding considerations have the force I consider them to possess, it follows that the lines which have been set to differentiate sclerema from xeroderma, and Scleroderma adultorum from S. neonatorum, can no longer be maintained, even as an aid to classification. The only question that remains is, the relationship that elephantiasis, leprosy, and tubercular diseases of the skin generally, bears to some cases of sclerema—such as that of Mr. Gaskoin's notably.

Since the essential feature of these affections is hypertrophy and induration of the subcutaneous connective tissue and skin, I think they must be included with sclerema in a general classification. With this view, I would define

¹ A comparison of the records of the cases included in the œdematous variety of sclerema (see p. 149 of this paper) with "Sclerema neonatorum" will prove the truth of this statement.

sclerema as an induration of the subcutaneous connective tissue, sooner or later in some degree involving the derm and epiderm, and causing proportionate alteration of structure and disturbance of function. Two or, perhaps, three classes may then be formed:—1. SIMPLE SCLEREMA, or induration without actual hypertrophy of the connective tissue or skin; 2. IDIOPATHIC HYPERTROPHIC SCLEREMA, in which the induration of the connective tissue is accompanied by positive hypertrophy; and 3. SPECIFIC HYPERTROPHIC SCLEREMA, in which may be included the tubercular disease of the skin.

1. SIMPLE SCLEREMA; under this may be grouped the following varieties:

(A) SIMPLE ATROPHIC SCLEREMA, marked by the gradual disappearance of blood-vessels, and shrinking of the connective tissue, so as to attach the corium, more or less completely, to the subjacent fasciæ, muscular aponeuroses, tendons, ligaments, or bones, and unattended by any previous changes in the parts affected.

This may be—(1) *General*, as in the case narrated, and in those given by Bruche,¹ Barton,² Day.³

(2) *Local*, as in the sclerema of the fingers and palm; in one of Mosler's⁴ cases (W. J.); in Case 3, related by Arnold,⁵ and further exemplified by Alderson's⁶ case; and in those cases of so-called Addison's keloid,⁷ which are simply atrophic.

(3) *Scattered*, which will include morphœa, and many cases of partial sclerema.

(B) ŒDEMATOUS SCLEREMA, such as occurs in children (Scleroderma neonatorum), in chlorotic girls, and those

¹ 'Hannov. Ann.,' vii, 5 and 6, 1847.

² 'Dub. Quart. Journ. Med. Sci.,' Aug., 1869.

³ 'American Journ. Med. Sci.,' April, 1870, vol. lix, p. 350.

⁴ Virchow's 'Archiv,' B. 23.

⁵ 'American Journ. Med. Sci.,' new ser., vol. lviii, p. 89.

⁶ 'Medico-Chirurgical Transactions,' vol. xxxvii.

⁷ *Ibid.*

in whom the catamenia are suddenly suppressed (see Thirial's¹ cases, Arnold's case,² one of Mosler's³ cases, two of Rilliet's⁴ cases, Fantonetti's⁵ case, and Sir W. Gull's two cases quoted by Fagge).⁶

(c) INFLAMMATORY SCLEREMA, including those cases in which the local affection has been preceded by some amount of inflammatory irritation. It may be—(a) *Acute*, as in the cases related by Henke,⁷ Bouchut,⁸ Gillette⁹ (which followed the application of a blister), Eckström (which followed an attack of erysipelas),¹⁰ Arning,¹¹ and by Fuchs.¹²

(b) *Chronic*, as in the cases given by Fiedler,¹³ Forget,¹⁴ M'Donnell,¹⁵ Nordt,¹⁶ in the very interesting and typical one by Mr. Gaskoin,¹⁷ and those by Sedgwick¹⁸ (partial) Mosler (in one), perhaps that by Grisolle,¹⁹ and that by Förster.²⁰

(d) TRAUMATIC, following upon injuries, such as varicellous and syphilitic inflammations, burns, the use of the cat, and ulceration.

¹ 'Gaz. Méd. de Paris,' 1845, p. 523.

² 'Amer. Journ. Med. Sci.,' new ser., vol. lviii, p. 89.

³ Virchow's 'Archiv,' B. 23.

⁴ 'Traité des Malad. des Enfants,' 1861, vol. ii, p. 107.

⁵ 'Annali Universi di Milano,' 1847.

⁶ 'Guy's Hosp. Rep.,' 3rd ser., vol. xiii, p. 286.

⁷ 'Handb. zur Erkenntniss und Heilung der Kinderkrankheiten,' 1809.

⁸ 'Gaz. Méd. de Paris,' 1847, p. 771.

⁹ 'Archives Gén. de Méd.,' 1854, tom. ii, p. 657.

¹⁰ 'Hygiea,' Band ii, No. 2.

¹¹ 'Hygiea,' Band ii, No. 2, and 'Würburger Med. Zeitschrift,' 1861, Bd. ii p. 186.

¹² 'Bericht. über die Med. Klin. zu Gottingen,' 1855, p. 192.

¹³ 'Deutsche Klinik,' 1855, p. 34.

¹⁴ 'Gaz. de Strasbourg,' No. 6, 1847.

¹⁵ 'Dub. Hosp. Gaz.,' 1855, vol. ii, p. 6.

¹⁶ Virch. 'Arch.,' 1861, vol. xxii, p. 198.

¹⁷ Page 113 of this volume.

¹⁸ 'Pathological Trans.,' vol. xii, 1861, p. 234, and vol. xvi, 1865, p. 260.

¹⁹ 'Gaz. des Hôp.,' 1847, p. 209.

²⁰ 'Würzburg Med. Zeitschrift,' 1861, Band ii, p. 294.

2. IDIOPATHIC HYPERTROPHIC SCLEREMA.—

That an increase of the connective tissue and of the skin can occur during the progress of simple atrophy of the integument is obviously impossible. Nor can I consider that actual hypertrophy has been proven in any of the recorded cases, for the reasons stated at pp. 142, 143, but that such a condition may arise in consequence of prolonged subacute inflammatory action so insidious as to exclude it from B and C, Class 1, is possible, and such cases would be properly included in this provisional class.

3. SPECIFIC HYPERTROPHIC SCLEREMA, including the following :

- (A) SYPHILITIC HYPERTROPHIES AND CONDYLOMATA.
- (B) ELEPHANTIASIS.
- (C) LEPRO ARABUM.
- (D) MOLLUSCUM.
- (E) KELOID (the tubercular as distinguished from the cicatricial form).

Such a classification as the above has the advantage of bringing into a single view a number of morbid conditions more or less closely allied, and each of which will, I believe, throw some light on the etiology and pathology of the others.

All classification is, of course, to some extent artificial ; and just as occurs in grouping animals or plants, so here we shall surely meet with cases which will serve as links between the classes above defined.

APPENDIX.—During the present summer (1877) there has been a slight increase of weakness, and the patient has occasionally been obliged to keep his bed two or three days in succession, and the hands have become dusker.

After a sojourn at Walton-on-Thames, the patient presented himself at the hospital on the 3rd of August, when the external shade temperature was about 80° Fahr.

He stated that he was no better, and showed extreme debility. He was perspiring freely; the hands were darker than I had ever seen them, the ends of the fingers being bluish-black and cold. Dry gangrene, indeed, seemed imminent, and the integument over the summit of each metacarpo-phalangeal knuckle of the right hand was occupied by a minute dark adherent scab, like those which formed on the tips of the two fingers, which have undergone slight gangrenous shortening.

The sclerema, however, is less for the first time during the progress of the disease. The integument can now be slightly pinched up over the backs of the proximal phalanges of the right hand, the integument of the neck has lost its brawny consistence, and the skin over the upper part of the chest has resumed its normal appearance and mobility. There is no amelioration of the cardiac and gastric symptoms; the poor patient is a wretched object, the neck being very thin, and the features drawn and expressionless, like those of a mummy.

ON THE MINUTE ANATOMY
OF
TWO CASES OF CARCINOMA OF THE BREAST
PRECEDED BY ECZEMA OF THE NIPPLE AND AREOLA.

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(Received January 3rd—Read January 23rd, 1877.)

IN a paper read during the last session I described the minute anatomy of two breasts which had been the seat of long-standing eczema of the nipple and areola.

The present paper contains a description of the minute anatomy of two cases of hard carcinoma of the breast, which were preceded by eczema of the nipple and areola. For the first of these cases I am indebted to Mr. Savory; for the second to Sir James Paget. My thanks are due both to Sir James Paget and Mr. Savory for the trouble they have taken to further the following research, and for the very kindly interest they have shown in it throughout.

Mr. Savory's patient (Case 1) was a lady, æt. 66, in whose breast a tumour had only been noticed for seven weeks. The appearance of the tumour was preceded by an eczematous condition of the nipple and areola, but only

by a very short interval, about a fortnight. There was no family history of cancer.

In consequence of the rapid growth of the tumour amputation was performed at an early stage of the disease in June, 1876.

When handed over for examination the general characters of the growth were those of a circumscribed hard carcinoma, about the size of a small egg, situated in the upper and inner portion of the breast, and reaching to within about half an inch of the areola. The intervening space between the tumour and the areola was occupied by slightly indurated fatty and fibrous tissue and glanducts. The nipple and areola were considerably thickened. The nipple was hardly so prominent as usual; its surface was ulcerated, and the ulceration was partly covered by a thick scab. The condition of nipple and areola was not nearly so bad as in the first two cases examined.

Sir James Paget's case (Case 2) was that of a lady, æt. 48, who had suffered from eczema of the nipple and areola of about three years' duration. The breast had remained apparently quite healthy until three or four months ago.

Amputation was performed in October, 1876, and the breast was almost immediately handed over for examination. The nipple was retracted, but unequally so, being much more drawn down on one side than on the other. Its surface was here and there rough and eroded. An eczematous patch occupied about one half of the areola, spreading on to the side and up to the free surface of the nipple. This patch was raised, indurated, uneven, and scabbed over. The remainder of the areola appeared thickened, but not ulcerated or scabbed over. The tissues immediately beneath the areola were rather more indurated than normal. About half an inch below the areola commenced marked induration of what had every appearance of infiltrated or disseminated hard carcinoma, very different in character from the form of cancer in Case 1, but quite as typical of another not uncommon

kind of cancer. This condition extended through the whole of a large breast, infiltrating unequally, forming here and there a hard nodule as large as a small nut, but nowhere producing a large circumscribed mass. A dilated duct was traced down for about one inch below the nipple into the cancerous disease.

The breasts were treated in the same manner as those previously examined. Sections were made from all parts of the tumour of the nipple, and of the intermediate tissues; and the order of these sections was carefully preserved, so that there could be no doubt as to the position from which any given section was taken. Some sections were made perpendicularly, some horizontally, in order to trace, as effectually as possible, the connection between diseased tissues, which could not, save by the greatest good fortune, be observed in any single section.

Stated as shortly as possible, the appearances observed were as follows:

Proliferation of the mucous layer of the epidermis of the nipple and areola, and infiltration of the cutis and subcutaneous tissue with small round cells.

Alteration and proliferation of the epithelium of some of the galactophorous ducts and small-cell infiltration of the surrounding tissue. Besides these diseased ducts several ducts were observed in Case 2 (Pl. VII, fig. 1) dilated, but having a tolerably normal lining of epithelium (fig. 2). In both cases, too, a number of normal ducts were discovered.

Enlargement of the acini, which were filled with epithelium.

Infiltration of the surrounding connective tissue with small cells (fig. 2).

All these changes are such as were described as existing in one or both of the two former cases. Further is now to be added:

Dipping down of the epidermis in Case 2 into the cutis, and the presence of a few cell-nests, giving to the

thickened portions of the areola and nipple the characters of epithelioma.

Much greater enlargement of the acini and ducts in the centre of the carcinoma than in the previous cases, so that they had frequently become confluent (the partition-walls being broken down), and their contents had made their way out into the surrounding tissues.

The cancerous change had, however, nowhere advanced so far in Case 1 (fig. 4), but that the rough structure of the breast could still be distinguished, although considerably altered.

This constitutes the rough description of the microscopical appearances. But, whereas formerly I was obliged to be contented with a mere description of appearances, I am now driven to express some opinion as to the origin of the carcinoma, and as to the connection which may exist between it and the eczematous condition of the nipple and areola.

With regard to the various views respecting the origin of carcinoma, the most important of them may be thus expressed—(i) That carcinoma is derived from the connective-tissue elements, (ii) that carcinoma is derived from the endothelium of the lymphatics, (iii) that carcinoma is derived from epithelial elements, (iv) that it is derived from the colourless blood-corpuscles.

In the present case I shall, I think, be able to prove that the carcinomata were derived from the epithelial elements of the mammary gland and its ducts; but, before attempting this, will endeavour to show that it could not have arisen from any of the other elements.

For such negative proofs Case 1 is particularly well adapted, as the various structures in the younger portions of the tumour are very distinct and well defined.

Looking very closely at the cell elements lying within the ducts and acini, and at those lying in the surrounding connective tissue anywhere, except in the most advanced portions of the carcinoma, so great a difference exists that it would be impossible to mistake them or to con-

found them together (figs. 2, 3, 5—9). The cells in the ducts and acini are of larger size, many of them of very much larger size than those lying outside. Their nuclei are also large, with well-marked outlines, having one, two, or more nuclei, not colouring very deeply with hæmatoxylin, showing generally a very granular appearance. With the exception of the borders of the cells or remains of the old cells, nothing in the ducts and acini has coloured deeply. Sometimes there is seen only a nucleated protoplasm (fig. 3), not differentiated into cells, like granulation tissue, but with large nuclei like those just described. Although there are great differences in size and shape, not only between the cells of different acini and ducts, but even between those of the same duct and acinus, such differences are easily reconcilable, and the gradual steps may be traced which connect the most distant forms. The cellular elements of the connective tissue are all easily classed under two forms—(1) small bodies, rather nuclei than cells, well-defined, equal in size, generally round or spherical, but often irregular in shape, not nucleolated, colouring *very* deeply, sometimes but not very often surrounded by a small quantity of protoplasm; (2) oval bodies of larger size, but not nearly so large as most of the nuclei of the cancer cells, not very vigorously defined, generally containing one, sometimes two nucleoli, colouring very faintly, and not shewing a granular appearance. These bodies were not nearly so numerous as the first.

For the purposes of discussion, the cells lying in the connective tissue, may be supposed to be derived from the connective-tissue elements, or from the blood-vessels. If the cells in the ducts and acini are formed from them, it must be by considerable changes. Traces of such changes ought to be discoverable either in the connective tissue, or in the ducts and acini, or in the walls of such ducts and acini. No one could for an instant imagine that these cells, wandering into the ducts and acini, became there suddenly transformed into the cells with which these

cavities are filled. I have looked most carefully in all three of these situations, but have totally failed to discover any such transitional forms. Occasionally one or two of the small dark corpuscles can be found in the midst of the larger cells in the acini, but quite unchanged, and apparently in no way affected by the neighbourhood of these cells. The presence of such a cell in the cavities only makes the difference in the characters of the cells more striking. Again, the position of the corpuscles in the connective tissue is not at all suggestive of continual influx into the ducts and acini, for they frequently lie at some distance from these cavities; and even where they lie in tolerably close proximity, the walls are almost always quite free from corpuscles. Against these facts, I know of nothing of equal value which could be set forth in favour of the connective-tissue or blood-corpuscle origin of the carcinoma. It might of course be argued, that in the earliest stages of the disease the cells within the ducts and acini are formed from the connective tissue or blood elements, and that transitional forms exist during that period. If that ground be taken, it must then be admitted that such a mode of formation has ceased, and is replaced by another totally different mode, so that either theory would thereby lose almost all its value. Against it, on the other hand, I have to set the same appearances as those described in the two breasts examined last year, in which the difference between the two sets of corpuscles was just as marked as in these breasts.

The same arguments serve equally well against the lymphatic origin of the disease, whilst to them may be added, that the first formations of new cells of the layer types are not observed in or near the positions assigned to the lymphatics of the breast.

To turn to proofs of the epithelial origin of the disease, I would point to the change in the characters of the epithelial cells lining the ducts, the transformation of cylindrical into spheroidal or squamous cells, the widening out of the ducts and complete filling up with these

cells; the dilatation of the withered acini or new development of acini now filled with the same kind of cells as fill the ducts; the complete absence of all other cells save the epithelium from which these new cells could be formed; the continuous dilatation until the cavities are fused together, their contents burst forth into the surrounding tissue, and cancer is fully formed; the evident youth of numbers of the cells, and the presence in some of the cavities of nucleated protoplasm. As to the manner in which the new cells are formed it appears to be sometimes by multiplication of nuclei and division, falling short of which the nucleated protoplasm is produced; or by endogenous multiplication (figs. 5—9), of which there are many evidences in most of the ducts and acini (fig. 2). The exact manner of this endogenous multiplication I have not been able satisfactorily to discover from these specimens.

If it be asked what is the relation which the cell elements in the connective tissue bear to those in the ducts and acini (which I should regard as essentially the cancer cells), I should say that they are the result of irritation, the product either of blood-corpuscles or connective corpuscles, or perhaps both. They have all the characters of such cells; they are found in great abundance in the close vicinity of the blood-vessels; similar cells can be seen in the blood-vessels. Such cell tissue is the natural result of irritation and inflammation. With respect to the theory lately put forward by Dr. Creighton, of the relation between these small bodies and the cells in the cavities, there does not appear to me any sufficient proof that they are the remains of old vacuolated cells on their way to or within the lymphatic channels. The only point in which the two things resemble one another is the depth to which they both colour. In other respects they are most different. The remains of the vacuolated cells vary very much in shape and size, are much more frequently triangular or quadrilateral with incurved sides than round or spherical, are generally

larger than the corpuscles in the connective tissue, and have an incomplete appearance when separated from the thin outlines with which they are usually in connection. Nor are there any transitional forms to be found between the two, although they were just as carefully searched for as the transitional forms between the cells outside and inside the cavities.

From these and the two former cases the following conclusions may, I think, fairly be drawn :

(i) That a certain relation existed between the eczema of the nipple and areola and the carcinoma of the breast.

(ii) That one of the first effects of the eczema was to produce proliferation of the mucous layer of the epidermis of the parts affected.

(iii) That in time the epithelium lining the galactophorous ducts became affected in like manner.

(iv) That the disease travelling along the large ducts reached the smaller ducts and acini, which became dilated and filled with proliferating epithelium, which was at length, so to speak, discharged into the surrounding tissues.

(v) That the carcinoma thus formed was therefore essentially a disease of epithelium.

DESCRIPTION OF PLATE VII.

Eczema and Carcinoma.

Fig. 1. Horizontal section below nipple, showing disease in ducts. (B. 4 in.)

Fig. 2. Portion of duct with proliferating epithelium wall, and surrounding tissue containing corpuscles of two kinds. (Oc. 3, obj. 7, tube drawn out, $\times 260$.)

Fig. 3. To show duct or acini filled with epithelium (a good example of nucleated protoplasm). (Oc. 3, obj. 7, t. d. o. $\times 260$.)

Fig. 4. Sketches of portions of breast affected with carcinoma, showing structure of mammary gland remaining, but altered. (B. 1 in.)

Figs. 5 to 9. To show endogenous formation of epithelial cells. ($\times 260$.)

FIG. 1



FIG. 2

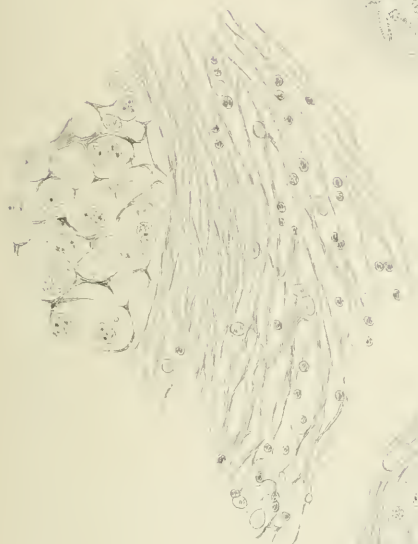


FIG. 4

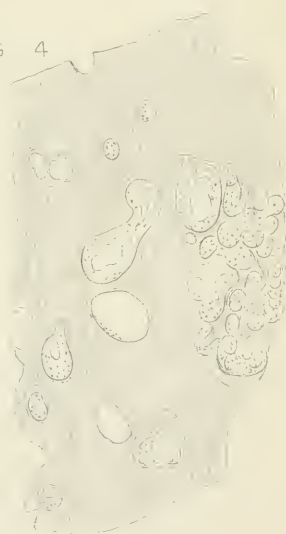


FIG. 3

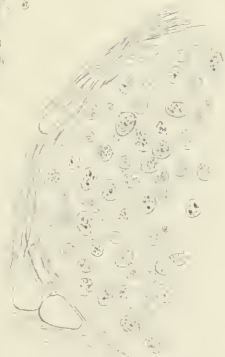


FIG. 5



FIG. 7



FIG. 8



FIG. 6



FIG. 9



DISLOCATIONS OF THE THIGH:

THEIR

MODE OF OCCURRENCE AS INDICATED BY EXPERIMENTS
AND THE ANATOMY OF THE HIP-JOINT.

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RECENTLY, whilst making various dissections of the hip-joint, the usually accepted doctrine that dorsal and ischiatic dislocations of the femur occur when the thigh is in a position of adduction appeared to me as very improbable, on anatomical grounds alone, and I was, therefore, led to test the truth of it by means of experiments on the dead body.

I propose in the following remarks to prove by anatomy, experimental results, and clinical facts—(1) that all kinds of dislocations at the hip-joint *can* take place while the thigh is abducted; (2) to give reasons for believing that abduction is the position in which all dislocations of the thigh happen; and (3) to show that in any given case the dislocation will be backwards, downwards, or forwards, according as flexion with rotation

inwards, or extension, or extension with rotation outwards, is associated with abduction at the moment of accident; or is provoked by the same violence which produces the displacement.

First, I will state briefly the anatomical features which bear upon the question.

The *acetabulum* looks forwards as well as outwards and downwards, and receives the head of the femur, which also looks forwards. The thickest and strongest part of the innominate bone is that which forms the upper and posterior wall of the acetabulum; it is this part of the bone which enters into the formation of the weight-bearing arches of the pelvis, viz. of the pelvic brim, along which the weight is transmitted to the heads of the thigh bones in the upright and stooping positions, and of the vertical or ischial arch, along which the weight is transmitted to the tuberosity of the ischium in the sitting posture. This same part of the innominate bone forms by far the deepest portion of the acetabulum, the articular surface of which is fully an inch wide in its iliac portion, and nearly as wide in the ischial. Along the lower part of the acetabulum, on the other hand, the articular facet, so far as it exists at all, is very narrow; while the cotyloid notch intercepts the rim for nearly one inch.

The *head of the femur* presents a much larger articular surface above the transverse plane through its centre than below it. The measurement (in a horizontal plane), from a vertical line skirting the dimple for the round ligament to the outer limit of the articular surface on the *upper* aspect of an average femur, is an inch and two thirds; whereas to the outer border of the articular surface *below* is only three eighths of an inch. The notch for the round ligament is in the lower and posterior quarter of the head; the most prominent point in the head is below this notch, while the part above slopes off from within outwards. As a consequence of all this, when the thigh is flexed and adducted, the most projecting part of the head

passes into the deepest part of the acetabulum and presses against the broadest and strongest part of its wall; whereas in abduction this prominent part of the head of the femur projects beyond the lower part of the socket at the cotyloid notch.

THE LIGAMENTS.—It is only necessary to refer to two of the four ligaments of the joint, viz. the capsular and the round ligament.

The capsular ligament is large and loose, so that in every position of the limb some portion of it is relaxed. In thickness and strength it varies greatly in different parts; thus if two lines be drawn, one from the anterior inferior iliac spine to the inner border of the femur near the small trochanter (Plate VIII, fig. 1), the other from the upper border of the tuber ischii to the digital fossa (Plate VIII, fig. 2), the ligament above and behind these lines, is very strong; whereas all below, except along the narrow pectineo-femoral band, is very thin and weak, sometimes permitting the head of the bone to be seen through it. There are three bands auxiliary to the capsule, or more correctly speaking, three portions of the capsule, which have received separate names, and which for the sake of description are called ligaments, although it would be erroneous to suppose that either of these bands or ligaments are structures separate from the capsule. The best known of them is the *ilio-femoral* ligament, a large triangular mass of coarse, well-marked fibres, the broadest end of which is at the femur (Plate VIII, fig. 3). Its fibres are somewhat accumulated both along the outer and inner sides of the femoral attachment, while several curved intercolumnar fibres, which spring from the centre of the capsule, pass to the femur with the inner set.

This arrangement of the fibres has led to the ligament being called by some the "inverted Y-shaped ligament," and exaggerated representations of it, in accordance with this name, have been drawn. Such, for instance, are the diagrams of the ligament in Bigelow's work and in the works of those who have copied therefrom. The name is,

however, inappropriate, for although the double accumulation of fibres may be made, by dissection, to show somewhat the appearance of an inverted **Y**, there is not any natural interval between the two imaginary arms of the ligament beyond a small aperture for the entrance of a branch of the external circumflex artery into the joint. Except for this small opening, the strong central fibres which take a straight course to the femur are not interrupted, and are of considerable thickness. The mistake has arisen, I imagine, from the presence of two well-marked bands connected with the surface of the capsule along the upper and outer border of the ilio-femoral ligament. One of these, the shorter and posterior, passes to the tendon of the gluteus minimus from the middle of the capsule; the other, not hitherto described, but generally well marked, extends from the vastus externus to the long head of the rectus femoris, and moves with both these tendons (Plate VIII, fig. 3). The latter especially gives the appearance of a superadded outer arm to the ilio-femoral ligament, while the more oblique direction of the inner fibres completes the supposed **Y**-shaped outline.

The *ischio-femoral* ligament extends from the ischial border of the acetabulum to the upper and back part of the neck of the femur, just internal to the highest part of the digital fossa. When the thigh is flexed the fibres of this ligament pass almost straight to their femoral insertion, and are spread out uniformly over the head of the femur, but in extension they wind upwards and outwards to the femur in a zonular manner (Plate VIII, fig. 2), and form quite a thick roll along their lower border. The upper and back part of the capsule between the ilio-femoral and ischio-femoral ligaments is very strong and thick, although not so strong and thick as the ilio-femoral itself. The latter is as stout as the tendo Achillis, and is often more than a quarter of an inch in thickness; the rest of the strong part of the capsule varies down to one eighth of an inch or a little less in thickness.

The third auxiliary band is the *pectineo-femoral* liga-

ment (Plate VIII, fig. 3), a distinct but narrow set of fibres, extending from the pubis and pubic border of the cotyloid notch to the lower and back part of the neck of the femur just above the small trochanter.

The effect of these bands on movements of the hip-joint is as follows : The *ilio-femoral* supports the trunk in the erect posture by limiting extension ; it is made tense throughout in every position of extension, except when extension is combined with abduction, and then the outer fibres are relaxed.

The *ischio-femoral* band does not limit simple flexion, but is tight during flexion combined with adduction, and flexion combined with rotation inwards. In extension, in flexion with rotation outwards, and in abduction, it is quite loose. The part of the capsule between the ilio- and ischio-femoral ligaments assists the former in limiting simple extension ; it is also made tense in extension with *adduction*, but is most tense in *adduction* combined with slight flexion,—the stand-at-ease position.

The *pectineo-femoral* ligament is stretched in every position of flexion and of extension when *associated with abduction*. It is the structure which limits *abduction*, and is especially tight during abduction combined with slight flexion, and also when marked flexion with outward rotation accompanies *abduction*.

The *ligamentum teres*, so far as its attachments are concerned, needs no notice here, but its action and use require a word or two. Without discussing the differences of opinion which have been expressed on these points it will be sufficient to state, as the result of several examinations, that the ligament is most tight during *flexion combined with adduction and rotation outwards*, but that it is also tight, though to a less degree, *during flexion with rotation outwards*, as well as *during flexion with adduction*. As the thigh passes from a position of adduction with flexion to one of adduction with extension it becomes less and less tense, and is quite lax when full extension is

reached. In every position of *abduction* it is loose, while in *abduction* associated with simple flexion it is at its loosest, and the ligament is then folded upon itself so completely as to have its femoral and acetabular attachments on the same level and opposite to one another.

From a consideration of these anatomical facts, it is evident that no conditions of the joint can be more favorable to rupture of the capsule and dislocation of the thigh than those of *abduction*. In *abduction* the head of the femur is more than half out of the acetabulum, bulging over the transverse ligament; the cotyloid notch being filled with soft compressible fat allows the ligament to yield a little as the head of the bone is forced over it; and the most prominent part of the head of the femur is resting hard against the thin portion of the capsule. This part of the capsule is strengthened somewhat by the pectineo-femoral ligament it is true, but it derives no appreciable support from muscles as does the rest of the capsule. The ligamentum teres, being quite loose, is absolutely powerless to resist force, but is most favorably situated to snap under a sudden jerk. Finally, in *abduction* all the strong portion of the capsule is relaxed, excepting during complete extension, when the innermost fibres of the ilio-femoral ligament become tense. *In fact, it may be said that there is a natural tendency for the head of the femur to be displaced during abduction of the thigh.*

Nor is there any other position so favorable to dislocation as *abduction*. It is quite unnecessary to discuss the improbability of dislocation in positions of simple extension, when the ilio-femoral ligament is the resisting structure; it is only in the rarest, *i.e.* the so-called irregular dislocations, that this part of the capsule is ever ruptured, and even then only by the extension of the laceration commenced elsewhere, and the peeling off of the ligament from its bony connections.

There is, however, a pretty prevalent opinion that dorsal dislocations take place during adduction through a rent in the upper and back portion of the capsule, and that the

use of the round ligament is to resist forces which tend to drive the bone out in this direction. Now, as dorsal dislocations are more common than all the others put together, it is most likely that they occur through some strain upon the weak part of the capsule, and it is necessary to state the anatomical provisions against their occurring during *adduction*. It is only during *flexion with marked adduction*, and *flexion with rotation inwards*, that the head of the femur presses firmly against the upper and back part of the capsule, and even in these positions the most prominent part of the head is against the strongest and deepest side of the acetabulum. During *flexion with adduction* the ligamentum teres is on stretch, and therefore in action to resist displacing forces, so far as it ever can do so. The portion of the capsule against which the head of the bone is bulging is strengthened by various muscles, and these during adduction and inward rotation lie stretched over it; the most important of them is the obturator internus, which, when stretched, acts much like a ligament on account of the several tendons into which the muscular tissue is inserted and the connection of some of the ultimate fibres of these tendons with the bony origin of the muscle; the power of resistance of this muscle is increased too by the play of the tendons of the trochlear groove of the ischium, whereby the strain on the muscle is diminished. Finally, the close connection between the external rotator muscles, as they pass to their insertions, and also of the gluteus minimus, with the capsule, must strengthen and support the upper and back part of that ligament.

Thus, the anatomy of the joint, it seems to me, points to *abduction* as the *only position* in which dislocation of the thigh can possibly occur. Experiments upon the dead body confirm the conclusion suggested by anatomy. They also establish the fact that all the regular varieties of dislocation of the hip can be produced and reduced after the head of the femur has been displaced in *abduc-*

tion. It will be best at once to state that the only way in which I have been able to dislocate the thigh is by forcibly abducting it. I have tried, standing over the body, the pelvis of which has been firmly fixed by two men, to force the bone out of the acetabulum through the back of the capsule; I have removed all the muscles and tried the same thing when only the capsule and wall of acetabulum were left to resist, and when, by previously flexing and adducting, or flexing and rotating inwards the thigh, the strain upon the capsule is at its greatest. I have also tried, by bringing the legs across one another, and jerking and forcing the thigh with the whole leverage of the limb to assist me, as well as by striking with a heavy block the front and outer side of the knee, to dislocate the flexed and adducted thigh; but by none of these means have I ever once succeeded.

As soon, however, as the limb is abducted and jerking force is applied to it while in that position, the head of the femur bursts through the capsule, and the dimple for the round ligament can be felt beneath the skin of the perinæum. After the head has left the acetabulum it can be made to pass either towards the dorsum ilii or the ischiatic notch by *flexing and rotating the limb inwards* more or less; or on to the pubis by *extending and rotating it outwards*.

The following statements are based on *fifteen* experiments:

The results in all these cases were so similar that it seemed unnecessary to record the notes of more. Except when specially stated to the contrary all the dislocations were effected before any dissection whatever was commenced.

1. The rent in the capsule varied with the degree to which the upward displacements had been carried previous to dissection, but it was always limited to the thin portion of the capsule. In none did it involve the ilio-femoral or the ischio-femoral ligament, or the strong part of the capsule between them. In most cases it commenced at the pubis, ran obliquely downwards and backwards to the

neck of the femur, and then extended upwards to a greater or less distance towards the digital fossa, either peeling the capsule from the back of the neck of the femur or tearing it through at a short way from its attachment to that bone (Plate VIII, figs. 1, 2). In some cases the periosteum was torn away with the ligament from the pubic part of the acetabulum, and in one the whole of the under and inner side of the capsule was separated from the acetabular margin; but in all, the rent took the same general course along the femoral insertion of the capsule.

2. The ligamentum teres was ruptured in every case by the dislocating force, and not by the subsequent movements whereby the dorsal or other positions were obtained. This was ascertained by feeling for the dimple in the head through the perinæum. In all but three the ligament was detached from the femur, and with it, in some instances, a scale of articular cartilage. In three cases it was torn asunder, once near the femur and twice near its middle.

3. The pectineus, quadratus femoris, and external obturator muscles were ruptured, the two former in every case, the latter each time it was examined. The gemellus inferior was generally torn asunder.

4. The obturator internus was ruptured in two instances, and the adductors longus and brevis in one and the same case. In none was the neck of the femur embraced by the small rotator muscles (*viz.* gemelli, obturator internus, and pyriformis); nor could the head of the bone be made to pass upwards beneath the obturator internus tendon, *i. e.* between it and the capsule of the joint, but it could be forced upwards on to the dorsum over the posterior surface of the muscle.

5. In no case was the ilio-psoas or the gluteus minimus ruptured. In one of the cases in which the obturator internus was torn the pyriformis and the lower border of the gluteus medius were also lacerated.

6. In three cases the great sciatic nerve was lying in front of the neck of the bone, and was not seen in the dissection of the gluteal region until the dislocation was

reduced. In one case the nerve was carried before the neck and retained there after reduction.

7. In three cases the dislocation was compound,¹ so that the head of the femur protruded through the skin of the perinæum.

In order to ascertain if the head of the femur could be displaced through a "button-hole" opening of the capsule three experiments were made in the following manner:—A muscular flap was dissected up so as to expose the small rotator muscles and the sciatic nerve; these were next cut and the capsule was thus fairly exposed. An oblique incision was then made through the capsule along the line of the pyriformis muscle, reaching from the acetabular margin to the digital fossa, then by flexing the thigh to a right angle with the trunk and adducting it the head of the bone was displaced on to the dorsum ilii with very little trouble. But though the length of the incision varied, according to the size of the joint, in the three cases, from one and a quarter to two inches, in no instance did the head of the bone pass out of its socket without further rupturing the capsule to the extent of from seven eighths to one and three eighths of an inch further. The direction of the rent in each case was downwards from the incision, through the back of the capsule at a short distance from its attachment to the femur. Thus, the head of the femur escaped through no "button-hole" opening at the back of the capsule, but by tearing up a large flap in the back and lower portion of it.

These several experiments have convinced me of the

¹ Two of these three cases of compound dislocation occurred in subjects who had died of cancer of the uterus attended with great emaciation, the other in a tall, thin lad, æt. 17, who had died of diphtheria following immediately upon typhoid fever. No rigor mortis was present in either case, and it was thought at the time that no better subjects could be selected for obtaining dislocation by *adduction*. Yet, with three men holding the body steady, varied, prolonged, and very forcible efforts to dislocate in this manner failed; but as soon as the limb was abducted and a moderate degree of jerking force was applied, the head of the bone, in each instance, started through the skin of the perinæum.

importance, which Von Pitha and Bigelow have pointed out, of the ilio-femoral ligament in determining the characters of hip-joint dislocations, and that without a knowledge of this ligament we cannot understand the modes of their reduction by manipulation. But they also prove that more than the ilio-femoral portion of the capsule remains untorn; that, in fact, all the strong part of the capsule is uninjured, even when the rent extends along the back of it as high as the digital fossa. That this may be so will at once appear evident if the direction of the ischio-femoral fibres, which pass obliquely upwards and outwards to the upper border of the neck of the femur, is borne in mind. I cannot, however, agree with Bigelow in thinking that the obturator internus is either specially or "materially concerned in one variety of hip-joint dislocation." To say that the head of the femur when displaced towards the sciatic notch is below the tendon, but if on to the middle of the dorsum ilii is above it, is apt to convey an entirely wrong impression; while it affords no explanation whatever of the difference in the dislocations in question, or of their mode of occurrence.

It is not necessary, as I have proved, that the internal obturator should be ruptured in order to get a dislocation with all the appearances ascribed to the dorsal; and on the other hand, Bigelow's own drawing of a sciatic dislocation, or, as he calls it, "dorsal dislocation below the tendon," shows the head of the femur so clearly above the upper border of that muscle that it is a perversion of terms to describe it as a dislocation below the obturator internus. It is a dislocation above the tendon so far as the new position of the head of the bone is concerned, but it is, like all the other ordinary dislocations, below the tendon as regards the point of exit of the head of the femur from the acetabulum. If the acetabulum were fractured and the femur displaced directly on to the dorsum ilii, the tendon of the obturator internus muscle would not be necessary to support it there; the untorn part of the capsule and the upward pull of the muscles

would be quite sufficient for the purpose. But even muscular action is not required, for in the experiments on the dead subject and with all the posterior muscles cut, there was no tendency for the bone to slip from the dorsum into the sciatic notch. If, on the other hand, the head of the bone escapes at the lower part of the acetabulum, and reaches the dorsum by ploughing its way around the margin of that cavity, it may either tear through the obturator internus and gemelli, or mount over them, and in doing so drag up the obturator tendon.

The obturator, though it cannot prevent the head from rising above it, may undoubtedly oppose its further ascent on to the highest part of the dorsum, but so does the untorn part of the capsule, and the sciatic nerve may offer more effectual resistance to the upward passage of the femur than any of the small muscles at the back of the joint. I have seen the nerve not only checking the progress of the bone while these muscles were intact, but after they were all divided, so that the head of the femur could not be made to reach the dorsum ilii by employing any ordinary amount of violence.

What determines whether in any particular instance the dislocation shall be dorsal or ischiatic is *the degree of flexion and inward rotation* existing at the time of the accident or produced by the violence which dislocates the bone. If the *flexion and inward rotation* be *extreme*, the bone will plough its way more directly backwards, and less upwards, around the rim of the acetabulum, and *sciatic* dislocation will be the result. Similarly in the forward luxations, if *extension is associated with extreme outward rotation* the head of the femur will be thrown on to the symphysis pubis, whereas, if there is less rotation and more extension—hypertension, in fact—the femoral head will be drawn on to the upper border of the pubis. In the posterior dislocations the trunk is bent upon the thigh at the moment of luxation; rotation inwards gives the posterior direction to the head of the femur, while the extensors of the hip and the hamstrings

carry it backwards, and are assisted by the adductors in pulling it upwards towards the sciatic notch or dorsum ilii.

In the anterior dislocations the femur leaves the acetabulum during extension, rotation outwards gives the anterior direction to the head, while the ilio-femoral ligament, the ilio-psoas muscle, the tensor fasciæ, and the adductors, draw the bone upwards in front of the pubis.

Besides the *à priori* proofs of anatomy, and the results of experiments on the dead body, the nature of the accidents which cause dislocations of the hip, when carefully inquired into, support the view that they all occur when the thigh is *abducted*.

Dislocations, unlike fractures, arise from violence which strains by twisting rather than by direct force, hence the frequency of luxations of ball-and-socket joints. Almost every injury to a joint is accompanied by some twisting movement of the body upon the limb, or of the limb upon the body, produced either by the violence itself, or instinctively by the injured person.

Some of the positions of the body when such twists would act on the *abducted* thigh are the following: the limb advanced as in walking, running, or pushing; the extended attitudes of fencing and boxing; mounting a ladder, climbing a wall; kicking high; standing on one leg, with the other foot raised on a chair and the body inclined over the bent knee; sitting with knees apart as when one leg is thrown over the arm of an easy chair; sitting with one foot resting on the opposite thigh; stooping with the legs wide apart; crouching in the half-sitting half-kneeling posture; riding on horseback, when the horse falls, or, if he rears and comes over with the rider; the sudden turning of the body by a heavy crane; striking out while swimming; striding from one steamboat to another just as they are "yawning" apart. That these and such like have been the positions in which dorsal as well as other dislocations have happened the reports of many cases show; yet while it is universally

admitted that the forward dislocations occur during *abduction*, the posterior are always stated to happen during *adduction*. I feel sure, however, that it will be found, on carefully inquiring as to, and interpreting the description given of, the accidents which produce dorsal dislocations, that the thigh was *abducted* at the time. Since making the foregoing experiments, a case of dorsal dislocation has come under my care which justifies this statement, and is worthy of notice on account of the age of the patient. An intelligent boy of 7 years was admitted into the Middlesex Hospital on December 20th, 1876, with dorsal dislocation of the right thigh, and one and a half inch shortening of the right extremity. He was at once placed under chloroform and reduction was attempted, but without success, by the house-surgeon. On my arrival I was told that *nothing could be made out as to the position of the limb at the time of the accident*, but on questioning the boy I ascertained the following account. He was walking along Union Street on the right hand side, and was in the act of crossing over to the left. When his left foot was in the roadway and the right on the curbstone, the right slipped backwards from under him; he then fell upon the front of his right knee with his body inclined forwards and to the right side, so that he struck the ground with his right arm.

Interpreted, this means that while his legs were abducted (in the act of walking) the right became forcibly more abducted by his foot slipping, and this caused the dislocation; then, as the pelvis was already rolled backwards on the right side (which corresponds to inward rotation of the right thigh) in the act of walking, and as moderate flexion and a little more inward rotation was produced by the inclination of the trunk, the *dorsal* direction was given to the head of the bone.

The age of the patient makes the case a rare one. Mr. Holthouse in Holmes' 'System of Surgery,' states that only one out of fifty-one dislocations at the hip-joint happened in a person under ten years; Bryant refers to

one case, the youngest of fifty-four, in a child of six years ; and Erichsen, who remarks that "in children dislocation is rare, as the shaft will generally give way," gives one case of dorsal dislocation, in a boy of six years old. Sir Astley Cooper mentions one case in a girl of seven ; Mr. Powdrell reports a case of obturator displacement in a child six months old, in the 'Lancet' of May 16th, 1868 ; Hamilton refers to nine others below the age of eight years, which he had collected from various sources ; and Fayrer states that two out of twelve cases which came under his care in Calcutta were in children six years old, one a male and one a female. Reduction in my own case was effected by flexing the leg on the thigh, and the thigh on the pubis, circumducting outwards, then rotating outwards and bringing the ham at the same time down upon the table. As soon as the limb was rotated outwards the head returned with a very audible noise.

Finally, the great success which attends the methods of reducing by manipulation supports the idea that all dislocations occur during *abduction*, or what is the same thing differently stated, that the head of the bone escaped through the *lower part* of the capsule. In posterior dislocations, after flexing the thigh, the plan is to circumduct outwards, and rotate outwards ; and in anterior dislocations, after flexing the thigh, to abduct slightly and circumduct and rotate inwards strongly ; that is, in each set of cases reduction is effected, after first abducting the thigh, by rotating the head of the femur in the opposite direction to that in which it was rotated during dislocation, and in all cases the leg is flexed to relax the hamstring muscles and sciatic nerve. In this way the head of the bone is brought below the acetabulum and rotated back again into that cavity, not simply on the side where the acetabular margin is less prominent, *but where, also, a passage exists through the capsular ligament.*

If the adducted thigh had been displaced directly through an opening in the back of the capsule the

manipulation so successful in dorsal dislocations would be difficult and destructive; more of the capsule, and the small rotator muscles, including the quadratus femoris, would have to be broken down before the head could be guided round the acetabular rim to the cotyloid notch, while the conversion of a dorsal or sciatic into a thyroid or pubic dislocation, often described as having happened without force, could not be effected without still further violence. Certainly, in such a case, the rule laid down by Hamilton for reducing by manipulation, viz., "to carry the limb, in the first trial, in those directions only in which it is found to move easily," could not be carried out by the *ordinary method*, for the mere statement of the rule implies that the head of the bone, by following an unimpeded course should be made to retrace the one along which it was displaced. Many of the methods of extension aim at the same object as manipulation, viz. to get the head of the bone below the cotyloid notch, where it will most readily return into its socket, not because the resistance of the rim is there less, but because this is the neighbourhood in which the capsule is torn.

In the experiments above quoted, after pushing the head of the femur through an incision in the upper and back part of the capsule, it was found easy to drag it back again over the rim of the cavity through the same opening in the capsule; so that the resistance offered to reduction by the border of the acetabulum, even at its most prominent part, is not very great. Tedious attempts to reduce dorsal dislocations by extension would seem to contradict this, but they do not actually do so, and for the following reason, that the resistance is due to a different cause. In the posterior dislocations the untorn ischio-femoral part of the capsule lies between the front of the femur and the acetabular margin, and has to be untwisted by manipulation, or torn away by violent extension, before the head of the bone can regain its socket.

Accurate descriptions of the pathology of hip-joint dislocations are much wanted: hitherto it has been seldom

if ever specified which part of the *back of the capsule* has been torn, viz. whether it was the thick acetabular or the thin femoral portion.

The femoral part of the back of the capsule is ruptured whenever the dislocation happens during *abduction*; but if dislocation ever occurs during *adduction* the strong pelvic part of the ligament, which receives the strain of the head of the femur in that position, must either be ruptured or torn away from its attachment, so as to allow of the thigh-bone being displaced *directly* on to the dorsum ilii or behind the acetabulum—an accident which, I believe, is always complicated by fracture at the joint and especially of the rim of the acetabulum.

A case of this sort, in which both the head of the femur and the rim of the acetabulum were fractured, has been described and figured by Mr. Birkett in the fifty-second volume of the 'Medico-Chirurgical Transactions.'

The inferences to be drawn from the preceding remarks may be stated to be the following :

1. That the ilio-femoral ligament is a thickened triangular or fan-shaped area of the capsule of the hip-joint, but not a Y-shaped ligament; nor is it a structure distinct or separate from the rest of the capsule.

2. That the thickened portion of the capsule determines the kind of manipulation necessary for reduction, and that the ilio-femoral ligament especially ought to be relaxed by flexion of the thigh in attempting reduction, either by manipulation or by extension.

3. That the degree of flexion or extension, and of inward or outward rotation of the thigh at the time of luxation; and, subsequently, the bridling of the thick portion of the capsule of the hip-joint, determine the situation of a dislocation.

4. That the anatomical construction of the hip as well as the results of experiments show that dislocations of the thigh, *posterior as well as anterior* (uncomplicated by fracture at the hip-joint), always occur while the limb is in a state of abduction.

5. That the *posterior* dislocations result when flexion and rotation inwards accompany abduction, and the *anterior* when extension with rotation outwards accompany abduction, while the downward or thyroid variety occurs during abduction unaccompanied by rotation.

6. That by the movements of successful manipulation the head of the femur is reduced through the same opening through which it was dislocated, and that the head is thus brought by flexion, abduction, and *reverse* rotation, to this opening, which is at the lower and inner side of the joint.

7. That the new position of the head of the bone in sciatic dislocations, as in dorsal dislocations, is above the obturator internus, although in both varieties the head of the femur leaves the acetabulum through a rupture in the capsule below that muscle, and that for these reasons the division of the posterior dislocations into "dorsal above" and "dorsal below" the obturator internus is misleading, if not invariably incorrect.

8. That dislocation through a "button hole" is *not possible*—owing to the inelasticity of the capsule, and the large size of the head of the femur as compared with the width of the capsule—and that in the reputed cases of impossible reduction on this account the obstacle to reduction has really been either a portion of muscle or of the capsule itself, which has been carried before the head of the femur into the acetabulum; or a fragment of the head of the femur left in the acetabulum.

9. That the rim of the acetabulum of itself offers no real resistance to reduction.

10. That in the exceptional case of a *direct* dorsal dislocation the muscles and untorn capsule would resist reduction by manipulation as usually performed, and that this resistance would be appreciated by the surgeon.

11. That direct dorsal dislocations can only be produced by immense violence and are always associated with fracture of the acetabulum, or of the head of the femur, or of both.

12. That violent pain in dislocations at the hip-joint is caused by the sciatic nerve being pressed upon, or looped up by the femur, and that pain or paralysis after reduction is due to the nerve being dragged forward upon the neck of the femur, or lacerated in the act of reduction.

13. That in reducing such cases it would be well to lift the head of the bone away from the os innominatum, during the movements of flexion and abduction, so as to disengage the bone from the sciatic nerve.

In conclusion, I feel it due to myself to state that the above inquiry was not commenced with a view to publication, but merely to satisfy myself upon certain points, the working out of which, I have been led to hope, may interest others. I wish also to acknowledge my thanks to my friends, Mr. Lyell and Dr. Charnley, for their valuable assistance in the performance of the experiments; and to Mr. F. Steele, for the great trouble he has taken in making the drawings which accompany the paper.

APPENDIX.

July 5th 1877.—I have to offer my thanks to the Council of the Royal Medical and Chirurgical Society, for allowing me the opportunity of strengthening the arguments advanced in the preceding paper on “Dislocations of the Thigh” by publishing, as an appendix thereto, the following case and the accompanying illustrations (see Plate IX).

“Dorsal dislocation of the head of the femur.”

Between one and two o'clock in the early morning of June 20th, 1877, a hansom cabman, aged 29 years, was running on the *near* side of his horse, which was “bolting” east-

ward along Oxford Street. The man was endeavouring to stop the horse by tugging at the bit, but the horse, swerving to the near side of the road, got on to the pavement, ran against a shop window, and crushed the man between the cab and the shutters, seven or eight of which were torn down.

A police constable, who saw the accident throughout, and from whom I obtained the above account of it, says that as he approached the spot the man was lying on his back, motionless and groaning, and was dead before he could be taken to the hospital. The dislocation was first noticed in the post-mortem room, and no attempt whatever was made to reduce it previous to dissection.

At 2 p.m. on June 20th, that is, a little more than twelve hours after the accident, Dr. Finlay performed the post-mortem examination and from his report I extracted the following notes:—"There was a moderate degree of rigidity. The body was muscular and well nourished.

"The diaphragm was found to be ruptured in its tendinous part; behind and to the left of the pericardium the rent was transverse, four inches in length, and large enough to admit the hand. The left lung was compressed upwards, and the heart displaced to the right, so that its apex lay just beneath the sternum; the lower part of the left thorax was occupied by the stomach, a portion of the omentum, and about nine inches of the colon. The fourth left rib was fractured just outside its angle, and the seventh, eighth, ninth, and tenth ribs were fractured about two inches further forward. The right lung was adherent to the chest wall and to the diaphragm throughout its whole extent, its substance was engorged and œdematous. The heart was healthy, and contained a little fluid blood; the left ventricle was firmly contracted. The peritoneal cavity contained several ounces of bloody fluid. The spleen was ruptured at its upper part. There was a considerable quantity of blood effused into the loose cellulo-fatty tissue around the left kidney. The kidneys were healthy; the liver large and fatty.

“The right lower limb presented all the appearances of a dorsal dislocation of the head of the femur. On the outer aspect of the right thigh, about four inches above the knee, was a superficial bruise, extending in an oblique direction from above downwards and across; it was about six inches in length, and one inch and a half in breadth. There was a similar bruise about five inches below the knee.”

The following is a description of the dislocation as seen upon dissection. For this I am myself responsible. When the *gluteus maximus* was reflected the head of the bone was found uncovered by its capsular ligament lying behind the acetabulum and between it and the great sciatic foramen. From the *os innominatum* and the capsular ligament the head was separated by the *gemelli*, *obturator internus*, and part of the *pyriformis*, all of which retained their normal cellular and fibrous connection with the capsule. The gluteal muscles were uninjured.

The *quadratus femoris* had sustained only slight injury, a few fibres at the upper border alone being lacerated. The *obturator externus* was not divided, though many of the muscular fibres near their insertion into the tendon were ruptured; the tendon was tightly stretched over the back of the neck of the femur.

The *gemellus inferior* was completely ruptured and much contused, and so also were a few of the lower fibres of the *obturator internus*.

The tendon of the latter was tightly stretched beneath (*i. e.* in front of) the displaced head of femur.

The *gemellus superior* was ruptured and contused. The *pyriformis* was considerably damaged, a good half or more of the muscular fibres being divided; it was chiefly the lower half of the muscles which had suffered, but some few of its upper fibres were also torn.

The sciatic nerve was not involved.

The capsule was ruptured on its lower and inner side, and was cleanly peeled up from off the back of the neck of the femur as far as the digital fossa. The rent commenced

below the pectineo-femoral band, midway between the acetabulum and the femur, and ran (1) outwards and backwards to the neck of the latter, which it reached just above and behind the small trochanter, and (2) inwards and backwards across the thin portion of the capsule towards the acetabulum, which it nearly reached a little behind the ischial border of the cotyloid notch. It thus formed two sides of a large opening, which was made quadrilateral in form by the detachment of the flap from the back of the femoral neck (*vide* Plate IX, fig. 2).

There was a comminuted fracture of the acetabulum, and an oblique fracture of the ramus of the ischium. The fracture through the acetabulum extended along the line of union of the pubis with the ilium, and in addition, a triangular-shaped piece of the floor of the cavity, passing inwards from the cotyloid notch, was quite detached though not displaced.

The cotyloid cartilage, together with the thin adjacent portion of the capsule upon which rests the ilio-psoas muscle was torn away from the pubic portion of the rim.

The direction of the fracture of the ramus of the ischium was obliquely backwards and inwards, immediately in front of the tuberosity. A small quantity of blood was effused along the sartorius and rectus muscles below the level of the great trochanter.

Remarks.—The laceration of the capsule and the mode of occurrence of dorsal dislocations described in the foregoing paper are illustrated completely by this case.

The rent is confined entirely to the thin portion of the capsular ligament. The capsule is torn through at the lower and inner side of the joint, and a loose flap is formed by the peeling off of the thin posterior portion from the back of the cervix femoris, just as I have stated was the case in the dislocations which I experimentally produced in the cadaver.

With regard to the accident I submit the following explanation as being consistent with facts and as probably correct. While running near the head of his horse and

on the *near* or left side of it the man was thrown with violence against the shutters almost at the same moment in which the horse first struck the window. In this way his trunk was jammed between the shutters and the horse, and the left ribs were fractured. At the same time the spleen and the diaphragm were ruptured and blood was effused about the left kidney. The contraction of the muscular fibres of the diaphragm during the few expiring efforts at breathing would have had the effect of opening the rent in the tendon of the diaphragm, and of thus allowing the passage upwards of some of the abdominal viscera during their compression in inspiration. So far the injuries received were all on the left side of the body, but now the horse staggered onwards and drew the left wheel of the cab against the right side of the pelvis and the upper end of the right thigh, and in so doing forced the head of the right femur downwards and inwards through the inner and lower part of the capsule, and at the same time fractured the right side of the pelvis. As the right leg was stretched outwards (*i. e.* abducted) in front of the advancing wheel, the wheel made a partial revolution against the limb and produced the superficial abrasion on the outer aspect of the thigh, which, be it noticed, Dr. Finlay described as "extending in an oblique direction from above, downwards, and across." In the same manner the thigh was pressed forwards and turned inwards, whereby the head of the femur was directed backwards. This done, the wheel next revolved against the right limb below the knee and caused the second bruise referred to in Dr. Finlay's report, and increased the displacement of the limb. Meanwhile the man's trunk was gradually falling backwards as his thigh was being pushed forwards from under him and rotated inwards. Thus, can we at once account for the characters of the dislocation, for the position of abduction during which it occurred, and for the position of the body after the accident.

The dissection suggested some of the difficulties which may attend the efforts at reduction by manipulation.

If the obturator externus be not ruptured by the dislocating force, the tendon of that muscle being tightly stretched over the back of the femoral neck will be likely to act as a strap upon the bone, which must be relaxed by strong rotation outwards before reduction can be obtained.

Again, when the acetabulum is fractured, the want of resistance in the fractured part might make it very difficult to accomplish reduction, more especially when, as in this instance, both pubis and ischium are broken. Though no serious effort was made to reduce the femur, because of the rigor mortis and of the desire to complete the dissection without disturbing the relation of parts, yet from one very gentle attempt I am led to think that the mobility of the fractured bone during manipulation would possibly have interfered with the immediate success of the process. The difficulty, in all probability, would have been overcome by getting an assistant to hold the pelvis firmly between his hands, while the surgeon manipulated the limb.

In conclusion, I should like to emphasize the fact that no attempt whatever had been made at reduction in this case, so that dissection revealed to us the exact condition of things as they were caused by the accident and by it alone. It has been urged against my paper that experiments on the dead subject cannot be made the complete test of the mode of occurrence of dislocations in the living subject, and the justice of this I admit. But the rarity of opportunities of examining dislocations of the thigh as the result of accident, is so great that almost our only means of testing opinion is by experiment. Some idea of this rarity may be conveyed by stating that neither the Hunterian Museum nor the museums of King's College, University College, St. Thomas's, Charing Cross, St. George's, the Westminster, or the London Hospital, contain a single specimen of recent dislocation of the head of the femur, and that the only specimens in all the public museums of the metropolis are *one* at the Middlesex Hospital, *two* at Guy's, and four at St. Bartholomew's.

The specimen of the case here described has been presented to the museum of the College of Surgeons.

I have again to thank Mr. Frank Steele for his drawings, from which the lithographs were taken ; and my colleague, Mr. Nunn, who, being surgeon of the week when the case was brought to the hospital, kindly gave me permission to make what use I desired of the preparation.

DESCRIPTION OF PLATES VIII AND IX.

Dislocations of the Thigh; their mode of occurrence. By HENRY MORRIS, M.A., M.B., F.R.C.S.

PLATE VIII.

- Fig. 1. The capsule ruptured in dislocation. Front.
2. Ditto. Back.
3. Front of capsule, showing (*a*) the ilio-femoral ligament and (*b*) pectineo-femoral ligament.

PLATE IX.

- Fig. 1. Recent case of dorsal dislocation of the head of the femur.
a. Pyriformis. *b*. Obturator externus. *c*. Obturator internus and gemelli, passing beneath head and neck of femur.
Fig. 2. Rupture of capsule in the case of dorsal dislocation.
d. Femur flexed and abducted. *e*. Line of fracture between pubis and ilium. *f*. Symphysis pubis. *g*. Line of fracture of ischium.

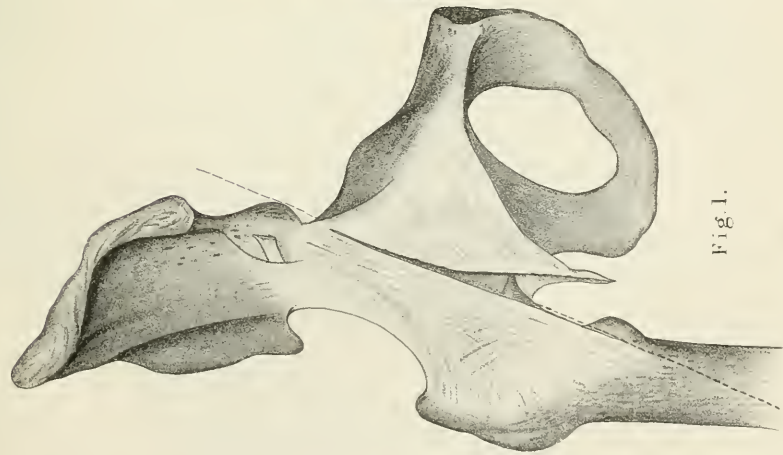


Fig. 1.

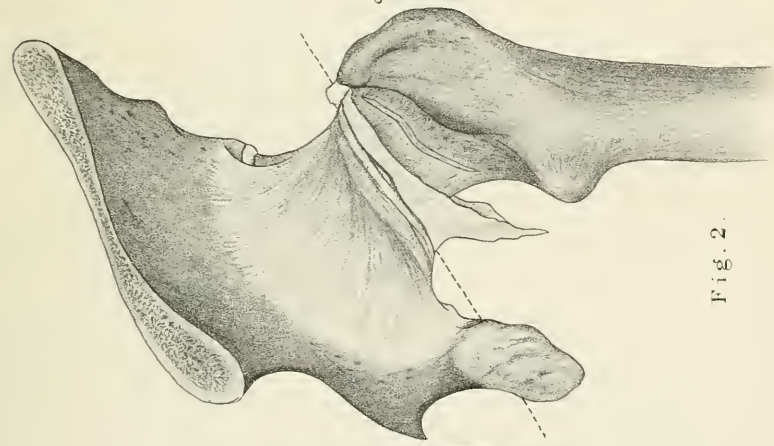


Fig. 2.

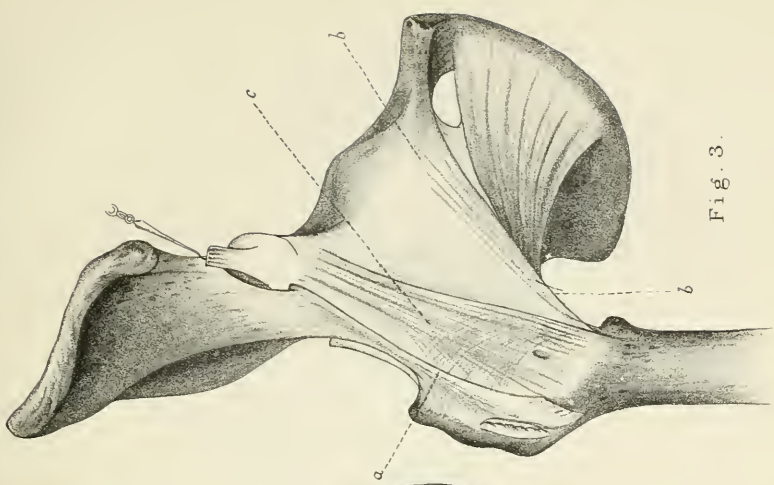


Fig. 3.

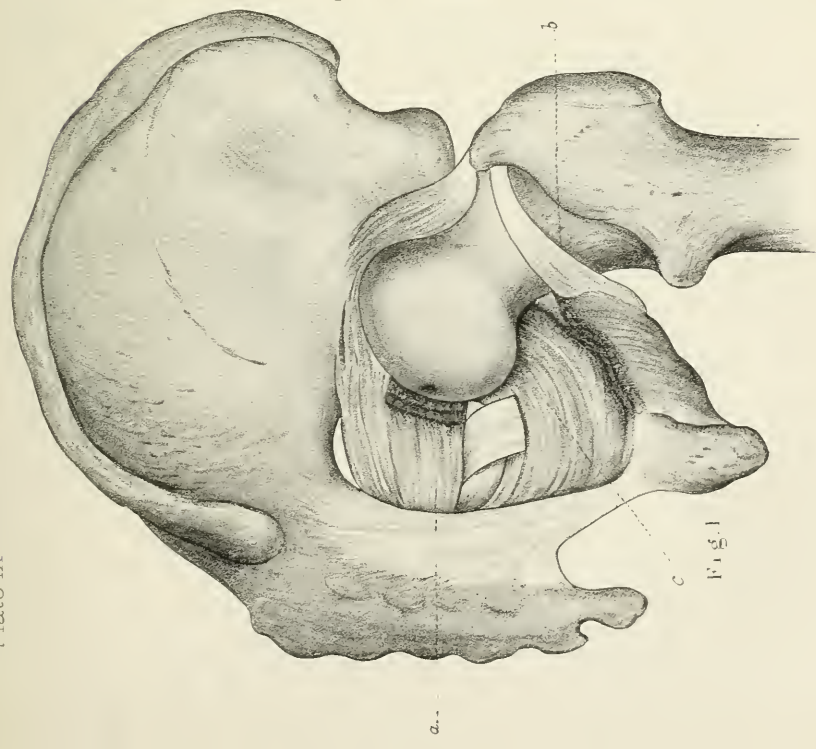


Fig. 1

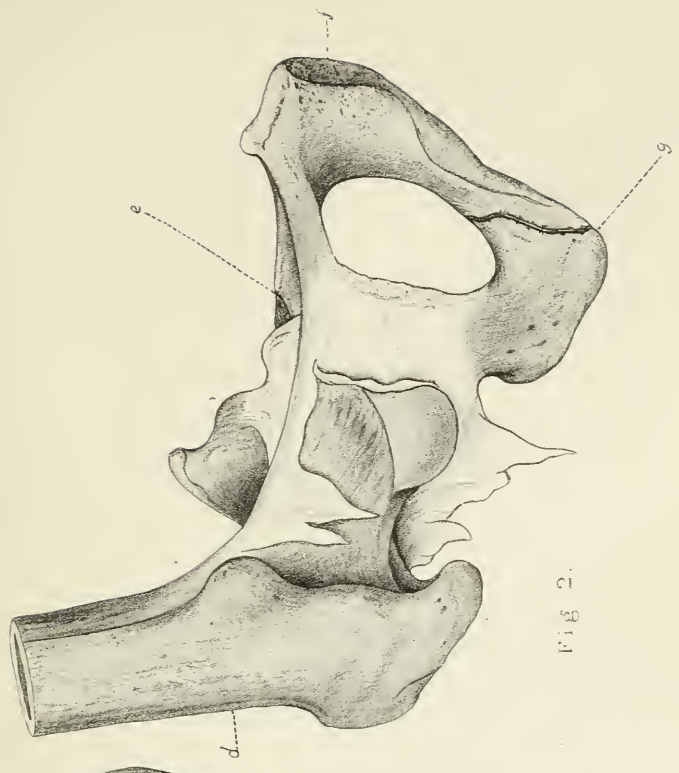


Fig. 2.

ON
NECROSIS WITHOUT SUPPURATION:

WITH
A CASE OF INTRA-OSSEOUS NECROSIS OF THE FEMUR
WITHOUT SUPPURATION, FOR WHICH AMPUTATION
AT THE HIP-JOINT WAS PERFORMED.

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(Received January 10th—Read February 27th, 1877.)

IN August, 1872, a man (J. B—), æt. 20, was admitted under my care, into St. Bartholomew's Hospital, at the request of my friend, Mr. Norton, whose patient he had been previously at the Holborn Union Infirmary.

The patient gave the following history:—He was quite well until about ten weeks before his admission into St. Bartholomew's Hospital, when he began to suffer from deep-seated pain, soon followed by slight swelling, in the left thigh. Both pain and swelling gradually increased, the pain being especially severe at night; and, shortly afterwards, being unable to work, he went into the Holborn Union.

For some time no fresh symptom appeared, and the swelling of the thigh, although increasing, assumed no

very definite character. About six weeks, however, after the beginning of his illness, as the patient was walking across the ward his leg suddenly gave way under him, and he fell, and on subsequent examination it was found that the left leg was much shortened, evidently in consequence of a fracture of the femur in its middle third. Some days afterwards I saw the case for the first time with Mr. Norton and Mr. Shepard, and the patient was transferred to St. Bartholomew's Hospital.

At this time there were the following symptoms:—The left lower limb, apparently shorter than its fellow by an inch or two, lay helplessly on its outer side, with the leg slightly flexed. The thigh was much swollen, and on careful manipulation, the swelling was found to be due to a tumour of the femur, hard and incompressible, which occupied the whole of the middle third, and extended also *below* to within a short distance from the knee-joint, and *above* to the trochanters. In its middle third, where the swelling was greatest, the femur looked deformed, and the existence of a fracture at this part was plain, although no distinct crepitus could be detected.

The integuments of the thigh were quite natural, and no abnormal condition of the soft parts between them and the tumour of the bone could be discovered. There was neither redness, nor œdema, nor elasticity as from confined fluid, but only the apparently unquestionable presence of a solid tumour involving nearly the whole femur, which at its thickest part had been fractured, and had thus led to shortening and deformity.

Great pain was caused by all manipulation, and still greater by any attempt to lift the limb.

The patient was exhausted and distressed from pain and sleeplessness, and he was said to have lost much flesh. His temperature was below 100° F. The femoral glands were not enlarged.

The case seemed, without any doubt, to be one of malignant tumour of the femur, which had become the seat of so-called spontaneous fracture, and with this

opinion my colleagues, several of whom kindly examined the case with me in consultation, entirely agreed. We were also unanimous in the belief that the best mode of relieving the patient lay in amputation at the hip-joint.

The patient readily assented, and as nothing appeared on a further general examination to forbid the procedure, I performed the operation by the commonly adopted method of cutting anterior and posterior flaps after transfixion of the limb.

Nothing unusual, or calling for special remark, occurred in the course of the operation. With the help of Lister's abdominal tourniquet there was less difficulty than I had anticipated in restraining the hæmorrhage, and altogether only a moderate amount of blood was lost. Space being allowed for drainage, the flaps, having been washed with a weak carbolic acid lotion, were united by silver sutures, and the wound was dressed in the ordinary way with carbolised oiled lint and cotton-wool.

For the first two days after the operation the patient was somewhat prostrate, but this was sufficiently accounted for by frequent vomiting, doubtless due to the chloroform.

On the fifth day after the operation the wound was suppurating freely. The temperature was 104.4° F. Pulse 120.

On the eighth day after the operation the temperature was 100.2° F., and from this date it gradually fell to the normal point. The wound began soon to heal rapidly, and excepting one part, at which a sinus remained and discharged freely for several weeks, the healing proceeded without interruption, and the patient left the hospital well.

On dissecting the limb, the condition of the integuments and muscles was found to be what had been previously anticipated. There was no inflammatory condition evident, no abscess or sinus, and not a drop of pus. On making, however, a longitudinal section of the femur and of the condensed tissue which formed its immediate covering, a very unexpected appearance presented itself. It was

plain that the disease from which the patient had suffered was necrosis of nearly the whole shaft of the bone. Where the femur had been fractured, the upper and lower fragments formed an obtuse angle. The broken surfaces were covered towards the inner aspect of the limb by fibrous tissue, forming a kind of false joint, but at the outer part the fracture was very irregular, and here the end of the sequestrum, rough and bare, projected from the upper fragment into a corresponding depression in the lower. The fracture was surrounded also by fibrous tissue in which a quantity of new bone had been deposited, so as to form a bossed and somewhat bulky callus. The bond of connection was, however, only feeble, and the callus was less apparent than it would otherwise have been, on account of its continuity with the new bone which had been deposited in and beneath the periosteum throughout the whole length of the shaft (Plate X, fig. 1).¹

The upper half of the femur, at its posterior aspect, had not perished with the rest of the shaft, but new bone had been deposited on this part, or rather, was growing from it and continuous with it.

As may be now seen in the specimen the dead bone is not at all points loosened from its connections. Near the trochanters it is still continuous with the cancellous tissue of the interior, and at the lower end of the bone, near the condyles, the same connection is observable. Between these two points, moreover, the medullary canal is encroached upon, more at the lower than the upper part, by new bone, which has been formed within the sequestrum, and which is continuous with the cancellous bone of the articular extremities and of the immediately adjacent portion of the shaft.

On the outer aspect of the sequestrum, dense and hard new bone has been everywhere laid down by the periosteum, so as to form a moderately thick sheath, which closely envelopes the dead bone.; so closely, indeed, that

¹ One half of the preparation is in the Museum of St. Bartholomew's Hospital, and the other in the Museum of the Royal College of Surgeons.

at some points it is difficult to make out any line of separation.

At other parts the space between the new and the dead bone is occupied by a thin but tough membrane, representing, I believe, the innermost layer of the periosteum.

Where the shaft has not perished in its whole thickness, the new bone and the old are continuous, so that, one might say, at these points there is *hypertrophy* instead of *necrosis*.

The periosteal sheath of new bone extends from just above the cartilage of the condyles to the trochanters, and around the whole circumference of the bone, excepting one or two small openings at which the dead bone lies exposed. But these openings look rather as if they were made accidentally after the removal of the limb than like cloacæ formed during life.

The chief interest of this case lies in the absence of suppuration, and the following questions naturally arise with respect to it:—Is the absence of suppuration in this particular instance exceptional? If it be so, what are the causes of the exception? If the freedom from suppuration be not exceptional how can the fact of its being usually unnoticed be accounted for?

The reply to these questions may be best given, I venture to think, in the following manner:

(1.) The case is one in which necrosis was the result of chronic inflammation of the bone, with incipient hypertrophy and sclerosis.

(2.) In such cases the necrosis presents many features which make it differ remarkably from the more ordinary kinds of necrosis, and especially from those in which the death of the bone has been caused by acute subperiosteal effusion, or by conditions which quickly lead to it.

(3.) In cases of necrosis occurring in the course of chronic osteitis, suppuration is not, as a rule, an early symptom.

(4.) The supposed rarity of the existence of necrosis

without suppuration may be due to the fact that opportunities of examining such cases, by section of the bone at an early stage, before suppuration has occurred, must be exceedingly rare, and, as in the present instance, the result of accident.

I will endeavour, as briefly as possible, to illustrate the preceding propositions, and state the grounds on which they rest.

I. *What are the special features which characterise necrosis occurring in the course of chronic ostitis?*

Time will not permit me to consider, in any detail, the pathology of inflammation of bone, but I may remark, in passing, that a careful examination of many specimens has led me to the belief that in the accounts usually given of chronic ostitis of a shaft, accompanied by hypertrophy and sclerosis, too much stress is laid upon the supposed *expansion* of the walls of the bone. Such expansion as exists is, I believe, only a textural expansion of Haversian canals, &c., by more or less absorption of their walls, without any simultaneous increase by any such process of the *girth* of the bone. The *hypertrophied* condition is rather one of overgrowth by the laying on of new bone both without and within, *i. e.* by both the periosteum and endosteum.

In most cases, probably, the hypertrophy thus produced ends as an hypertrophy, the new layers of bone becoming one with the original shaft. In other cases, however, the blood-supply of the shaft is gradually cut off, more or less, in the process of formation of the new layers; a process which may be readily conceived as very likely to starve a structure, such as the compact tissue of the shaft of a bone, which depends altogether for its supplies upon neighbouring tissues, viz. endosteum, periosteum, and the adjacent cancellous structure of the articular extremities. And especially may this be expected at a time at which

the blood-vessels of its own Haversian canals are more or less compressed by the products of inflammation.

The conditions which exist in a typical case of necrosis following inflammatory hypertrophy are thus characterised. (*a*) The necrosed bone bears evidence of having undergone certain structural changes before death. (*b*) It is very imperfectly separated from its connections. (*c*) It is more or less enclosed by new bone which has been produced by both the periosteum and endosteum. (*d*) The new bone is large, dense, and on its outer surface, stalactitic. (*e*) The conditions characteristic of complete necrosis, so well marked in one part of the bone, are replaced in other parts by conditions equally characteristic of chronic osteitis with hypertrophy and sclerosis; while, connecting the two, there is an intermediate or neutral territory, in which it is difficult to say whether the term necrosis or chronic osteitis is the more appropriate.

These characters are admirably shown in a specimen of necrosed femur from the Museum of St. Bartholomew's Hospital.¹ I have chosen it as an illustrative example, in preference to the recent specimen just described, on account of its greater age and, therefore, more marked characters; but the disease is the same in both (Plate X, fig. 2).

Perforated at various points by large cloacæ, it is plain that extensive suppuration must have occurred in connection with the necrosis; and, indeed, at first sight, the specimen seems a typical example of the ordinary cases of acute necrosis of the whole shaft of a bone. On further examination, however, it is evident that the specimen is one of necrosis, occurring in the course of chronic osteitis. The sequestrum is that of a bone which, before death, had undergone some of the structural changes characteristic of chronic inflammation. Its section is dense and almost ivory-like, while its outer and, to a less extent, its inner surface is rugged from irregular absorption and deposition of bone. Although of long standing, the sequestrum is in most parts immovable, and for various reasons. In the

¹ A. 95, Catalogue of Museum, St. Bartholomew's Hospital.

first place it is still in direct continuity with that portion of the shaft which has not perished, and there is no indication of even the commencement of a line of demarcation. Indeed, the only distinguishing mark is the appearance of the dead bone, which is whiter and smoother than the apparently living. The sequestrum is also riveted by new cancellous bone, which extends into its medullary canal for some little distance from the cancellous bone of the articular extremities. Here and there, moreover, through a 'break' in the sequestrum, *i. e.*, where the original shaft has not perished, the new bone without and within has become continuous, so as still further to lock the dead bone.

The periosteal sheath of new bone is separated from the necrosed part of the shaft by the usual groove, but as the sequestrum is continuous with the portion of the shaft which has not perished, so its sheath of new bone is continuous with that which is not so much a *sheath* as an *outgrowth* from the latter. In other words, the new periosteal bone (extending from one end of the shaft to the other), the section of the substance of which presents the same appearance in all parts, might be called a sheath at one part, and an outgrowth from the shaft at another. And inasmuch as there is scarcely any other distinction between the living and dead portions of the shaft it would be best, as a mere expression of the fact, to say, "where the new bone is separated by a groove from the original shaft the latter is dead; and where it is continuous with it, the shaft is hypertrophied."

It is worth noting that in this, as in the recent specimen, the upper and back part of the shaft of the bone is sclerosed and much enlarged, but not dead; while, suggesting a possible reason for this, there lies exposed, as it happens, by the section, the canal for a large nutrient artery, the calibre of which has not been interfered with. In both specimens, too, the necrosis is most extensive in the middle of the shaft, extending to the trochanter only along the anterior wall; while, on tracing the disease in

the opposite direction, it is found that, as the condyles are approached, the whole circumference of the bone escapes, and is hypertrophied merely.

On the outside of the specimen are the usual large stalactitic outgrowths of bone which form so characteristic a feature of chronic inflammatory hypertrophy, whether accompanied or not by necrosis within.

The contrast is great between the appearances which characterise this specimen and those which are observable in the more common instances of necrosis of a whole shaft. Take, for comparison, a case of necrosis which has been caused or at least accompanied by acute sub-periosteal effusion,—a case familiar to all. The question at once occurs to the mind, What is it which constitutes the difference? Is the distinction to be found in the *nature* of the disease, or in its *cause*? Or, is it in the *age* at which the disease occurs? for, it must be remembered, the sudden death of the whole shaft of a bone, by or with separation of its periosteum, is rarely or never seen, except in the young. I am disposed to believe that the age at which the two kinds of necrosis, here contrasted, takes place, offers the best solution of the difficulty, although it does not exclude others. A moment's consideration of the different anatomical conditions which exist in young and adult bones, respectively, will suffice to illustrate this. In a young bone there is but slight adhesion of its periosteum, and this is, therefore, readily stripped off by effusion of lymph or pus beneath. The shaft of such a bone, moreover, depends for its blood on two sources only of supply, viz. the periosteal vessels, and the nutrient arteries, both of which, by detachment of the periosteum, may be interfered with, and that completely; the epiphyseal lines of cartilage forming impassable boundaries, so far as anastomosis of blood-vessels is concerned, in a time of disease. Even if, as some suppose, the effusion beneath the periosteum be not the first condition which affects the nutrition of the bone, its occurrence at an early period of

the disease mostly ensures necrosis, when something short of sudden death may have otherwise ensued.

Hence it is, that acute necrosis of a whole shaft is most typically exemplified in the young. The supply of blood is quickly and completely cut off; the bone, of necessity, quickly dies, and the epiphysial lines form natural boundaries at which the dead bone is sharply and easily separated from the living.

At a later period, on the other hand, the anatomical conditions are changed. The union of the articular extremities with the shaft now supersedes the epiphysial line of cartilage, and thus a more complete system of anastomosing blood-vessels, continuous throughout the whole length of the bone, prevents any sudden necrosis, even if, super-added to the original inflammatory change, there occurs some separation of the periosteum by lymph or pus. The much firmer attachment, moreover, of the periosteum offers a greater obstacle to the tracking of fluid beneath it, and thus forestals one chief source of mischief. What wonder, then, if the necrosis be, so to speak, chronic, indistinct, incomplete; if the sequestrum be long in separating, shut up between layers of new-formed bone? The slow extension of the necrosis in these cases is by itself sufficient to account, in great part, for the long delay which occurs in the separation of the sequestrum. For it is plain that no line of demarcation can be formed in the case of mortification of any part, so long as the disease is still extending, and it would be difficult to imagine a case more likely to illustrate delay on this account than one of necrosis from chronic osteitis. Slowly, layer by layer, the new bone is deposited. Very gradually the old bone dies; and still the new bone increases. The original shaft is killed and buried by the same process. But no separation can occur so long as the line of junction of the living and dead creeps onward, and the longer the shedding of the sequestrum is put off, the thicker become the walls which imprison it.

II. *Suppuration is probably not an early symptom in cases of necrosis occurring in the course of chronic osteitis.*

That necrosis may occasionally occur without causing suppuration appears to have been long the subject of observation. Mr. Stanley seems to have considered such an event not unusual, In his work on 'Diseases of the Bones,'¹ he remarks, "When the bone attacked by necrosis is of small size, or only a small portion of it has perished, and in a patient who is not of an irritable habit, the inflammation which ensues in the surrounding soft parts is usually so mild that it gives rise to the effusion of fibrin or of serum, without suppuration; the disease then passing through its several stages unaccompanied by any other change than the simple enlargement, and thickening of the parts adjacent to the dead bone. Under such circumstances difficulty is often experienced in determining the nature of the disease, for it may then be doubtful whether the enlargement of the limb has been caused by necrosis, or by chronic inflammation of the bone, or by thickening of the periosteum, such as occurs in scrofulous children, in whom the periosteum of one bone, or of several, often becomes so much thickened as to produce an enlargement of the limb, simulating that which is caused by disease in the bone itself."

In the third volume of the 'Transactions of the Clinical Society,' Sir James Paget also calls attention to cases of necrosis without suppuration. He there narrates a case in which the symptoms were those of periostitis of the middle third of the femur, and in which the source of trouble was found to be a thin, rough sequestrum, about an inch and a quarter long, and a quarter of an inch wide, which lay in a flattened, irregular cavity, from which, on cutting through its outer wall, a little blood-coloured fluid escaped, and was followed by the protrusion of some soft

¹ 'A Treatise on Diseases of the Bones,' 1849, p. 83.

substance like coarse granulations. "The walls of the cavity, of which the outer was formed by the thickened periosteum, and the inner by the hollowed out surface of the femur, felt smooth and velvety, as if covered with granulations like those of ordinary cavities containing sequestra." Sir James Paget remarks, "the central point of interest in this case is, I think, in the fact of necrosis, leading to separation of bone, being unattended with inflammation of any of the textures external to the periosteum, or with more than a scarcely discernible amount of suppuration around the sequestrum. How unlike this is to the ordinary course of necrosis I need not describe." And he adds, that he had seen only one similar case, that of a boy, thirteen years old, in the upper part of whose left humerus was a large ovoid swelling, which had slowly and painfully increased for about a year. An exploratory incision having been made, the periosteum was found greatly thickened, and beneath it were some cavities containing thickened and half-dried pus, and several small sequestra from the wall of the humerus.

Sir James terms the disease, of which these cases are examples, *quiet necrosis*; and the epithet is equally applicable to the cases referred to by Mr. Stanley. The term, however, cannot be applied to a process which leads, with such symptoms as those which have been described, to necrosis of nearly the whole shaft of a femur; and it is I venture to think, to the *cause* of the necrosis, viz. chronic osteitis, rather than to the *quietude* of the diseased process, that we must look for an explanation of the absence of suppuration.

Granting however, it may be said, that the necrosis is the result of chronic inflammation of the bone, suppuration should be established when once a definite sequestrum is present. How can its absence be explained? The reply to this question may be best made, I believe, in the terms of the proposition, "suppuration is not of necessity an early symptom in cases of necrosis following chronic or subacute inflammation attended by hypertrophy and sele-

rosis;" and it may be added, suppuration may never occur at all.

This statement, so contrary to the generally accepted beliefs, does not rest on theoretical grounds alone, or on the recent case here related. As an illustration of its truth I am able to exhibit two very remarkable specimens from the museum of St. Bartholomew's Hospital¹ (Pl. XI, figs. 1, 2).

They are described in the catalogue as having belonged presumably, to the same person. One is the section of a femur, "in which a portion of the whole circumference of the inner layers of its wall, six inches long, has perished, and has been separated from the surrounding bone. New bone has been abundantly formed in and upon the outer layers of the wall, which is separated from the sequestrum. There is not in this new bone any aperture or cloaca, leading into the narrow space around the sequestrum. The tissue of the new bone is compact and heavy, and its surface hard and nearly smooth. It will be observed also, the description in the catalogue continues, "that the medullary tissue is entire within the perished part of the bone, although in this situation its tissue is nearly consolidated."

A. 119. "Section of a tibia, which is presumed to have belonged to the same person as the femur last described, but from the opposite limb. It is diseased in exactly the same manner. A sequestrum of a large portion of the inner layers of its wall is completely enclosed within the thick and hard layer of new bone formed on and united with the remaining portions of the wall. The exterior of this new bone is even smoother than that last described, and there is no aperture leading through it to the cavity containing the sequestrum. The medullary tissue is entire; but partially consolidated."²

¹ A. 118 and A. 119, Catalogue of Museum, St. Bartholomew's Hospital.

² These specimens are referred to by Mr. Stanley (*loc. cit.*), as the only example he had ever seen, in which *deep-seated* necrosis was unaccompanied by fistulous tracks in the bone and in the surrounding soft parts; but he does not suggest any explanation.

The close likeness which exists between the relations of the dead to the living bone in these specimens and in the two previously exhibited is remarkable ; and nobody can doubt for a moment that all are examples of the same kind of disease. There is the same continuity of dead and living bone, the same imprisonment of the sequestrum by new bone without and within, the same evidence of hypertrophy with sclerosis advancing side by side with necrosis, and the evident practical impossibility of removing the sequestrum by any surgical operation short of amputation. No history is attached to the specimens ; but, doubtless, the case was diagnosed during life as one of chronic inflammation with hypertrophy of bone. How many of the bones, it may be fairly asked, so diagnosed now, contain sequestra, greater or smaller, buried in them ?

I am indebted to Mr. Barwell for drawing my attention to a specimen of necrosis, almost entirely unaccompanied by suppuration, belonging to the Museum of Charing Cross Hospital ; and for permission to exhibit it to the Society. The specimen is a femur, the upper part of the shaft of which is occupied by a sequestrum, nearly three inches in length, imbedded in new bone which has been produced by both the periosteum and endosteum. The new bone on the outside is extremely thick and dense. During life, the disease closely simulated a slowly-growing malignant tumour.

The long delay which occurs in the separation of the sequestrum, in cases of necrosis from chronic osteitis, is probably one chief cause of the delayed suppuration.

The fact that dead bone, when not separated from the living, is less likely to excite suppuration than when it exists as a loose sequestrum, was noted by Mr. Stanley in the following passage.¹ "Instances of necrosis in early life have occurred, wherein a small portion of the dead bone, not separated from the living bone, has remained unchanged for many years, and the fistulous passages in

¹ Loc. cit., p. 116.

the soft parts leading to it have become closed." And again, after referring to the fact that, in such cases, a fresh attack of inflammation may ensue, with re-opening of the fistula, he adds, "The complete cicatrization of the soft parts over dead bone, however small its extent, is a rare occurrence, and it probably occurs only in the instances where the separation of the dead from the living bone has failed to take place. When the dead bone is detached from the living bone, it becomes as a foreign body, an irritant to the adjacent parts; purulent fluid is in consequence formed around it, the outlet for which is the fistulous passages in the surrounding soft parts." "But, on the other hand, a small piece of dead bone, retaining its connection with the living bone, may excite so little irritation in the surrounding parts, that no suppuration from them ensues, and, under such circumstances, it is not unlikely that the fistulous passages leading to the dead bone may become closed."

Mr. Stanley implies, here, that fistulous tracks are present at some stage or other of the disease; but the definite manner in which he directs attention to the comparatively small amount of irritation that is excited by unloosened dead bone, may be fairly taken as an additional support to the suggestion here made that, in the long-delayed separation of the dead from the living bone, may be found one great cause of the absence or long-delayed occurrence of suppuration, in cases of necrosis from chronic osteitis. But, after all, this is no exception to what is often seen in analogous instances of disease. Senile gangrene, for example, is not at once followed by suppuration. It is only when the dead part begins to be loosened at its line of junction with the living, that the process of suppuration is set up. Say that so many days or weeks are occupied in the complete separation of the skin and other soft parts, suppuration may not be noticeable until a third or a half of this time has elapsed. In the case of necrosis of the shaft of an

adult bone, from chronic osteitis, the separation may occupy many months or years. Is it, therefore, to be wondered at, that the delay in the occurrence of suppuration bears some sort of proportion? Its earlier occurrence would be not less useless than purposeless.

III. The peculiarity of the case which forms the text of this paper is to be looked for, I believe, rather in the rarity with which fracture occurs at an early stage of chronic osteitis with necrosis, than in the nature of the case itself. In other words, in the absence of the fracture, the disease would have been diagnosed as chronic inflammation with hypertrophy of the femur, with, perhaps, just so much suspicion of malignant disease as to lead to its being carefully watched, and, in time, explored, if the swelling increased. Had the pain subsided, and had no suppuration occurred, the patient might never have been operated on; and the existence of necrosis would probably never have been known. And if one considers all the circumstances which would interfere with the examination and preservation of the specimen, in the event of the patient not being operated on, it will not seem remarkable that specimens of necrosis without suppuration should be so rare. On the other hand, if suppuration had occurred, as it almost certainly would as soon as any small fragments of dead bone had become loose, the case would have been merely diagnosed as one of ordinary necrosis; and an operation, perhaps many more than one, would have been performed for its relief; and had the limb been amputated at last, after repeated failure of attempts to take away all the dead bone, or had the patient died from the effects of the disease, or of some other, the case would not have seemed remarkable; and although the bone might have been preserved in a museum, the special interest of the individual specimen to the surgeon would have ceased, as soon as a section of the limb had shown how futile must, of necessity, have been

all endeavours to remove the dead bone by any other method than amputation.

These considerations tempt one to foretell that section of such bones at an early stage of the disease, when the rare opportunity offers itself, will occasionally show the presence of necrosis without suppuration. Section at a later stage, after suppuration has occurred, will show a diseased condition, which, though long known and described, has not been definitely recognised as anything more than exceptionally long-delayed separation of the dead bone; whereas, if the suggestions now made be correct, the condition should be looked upon as *characteristic* of a special kind of necrosis, namely, of that which occurs in the course of chronic inflammation of a bone and its membranes, and more especially if the chronic inflammation take place after a certain *age*, namely, that at which the epiphyses become one with the shaft.

Reading the cases of "quiet necrosis," published by Sir James Paget, in the light of what has been shown by the present case, one may ask whether it is possible that, in these, deep-seated necrosis may have been present, and that the small outlying fragments which were within reach of a surgical operation once formed part of a sequestrum, the remainder of which, not yet separated, lay permanently entombed by new and sclerosed bone.

The imprisonment of a sequestrum by the formation of new bone on both its periosteal and endosteal aspects, for which condition I venture to propose the term *intra-osseous* necrosis, has not received the attention which it deserves either from a pathological or surgical point of view. For, apart from all theory, it is an evident indication of a diseased process which does not occur in the more common kinds of necrosis, in which no endosteal production of new bone takes place; while to the surgeon it is of interest, inasmuch as it may render the removal of a large sequestrum, by any means short of amputation, an impossibility.

The difference between *intra-osseous* necrosis, and the

more common kind of necrosis, in which so much may be done by an easy surgical operation, becomes evident when the two are compared. In one, taking for example a case in which death of the shaft of a bone and of its medulla has been a consequence of subperiosteal suppuration, there is a more or less hard shell of new bone, here and there perforated by cloacæ, and containing, as a loosely held kernel, the more or less rotten and hollow sequestrum. Even if not completely separated from its attachments, the dead shaft is held only by one or both of its extremities. In cases of *intra-osseous* necrosis, on the other hand, there is the hard shell of new bone as before, and within it the sequestrum; but the latter is not loosely contained or held only at its extremities. Within it, as well as without, is new and living bone, continuous, of course, with the living bone of the articular extremities, as well as with any portions of the shaft which have not perished; and through the latter with the periosteal new bone of the exterior. A transverse section best brings to mind the conditions present. Outside is the shell of periosteal new bone; next within is a more or less complete tube of dead bone; while innermost is the section of the solid endosteal rod of new bone, on which the dead bone is placed like a ferule. Even apart from the frequent absence at many points of the slightest line of demarcation between the dead and living bone, the complete removal of a large sequestrum, without destruction of the whole thickness of the bone is, it will be readily granted, impossible.

The bearing of these points on surgical operations, undertaken for the removal of necrosed bone is not, perhaps, often considered. There is no reason, indeed, for their dissuading from any attempt to give relief by operation, as it is impossible to say beforehand how much or how little of the circumference of a bone is affected; but there are, probably, few surgeons who cannot recall to mind cases, in which some such anatomical conditions as those here referred to may have been the cause of the

unexpected failure which attended long-continued endeavours to remove dead bone which had seemed well within reach.

The following are the conclusions to which the foregoing facts and arguments seem to lead :

1. That nearly the whole shaft of a long bone may perish, and that, nevertheless, suppuration, after several weeks, or months, and possibly, even years, may be still absent.

2. That necrosis of a long bone may, in the absence of suppuration, closely simulate malignant disease, even to the extent of undergoing so-called spontaneous fracture ; and that the latter event may not, for at least several weeks, be followed by suppuration.

3. That this apparently strange deviation from the course of the symptoms usually accompanying necrosis is probably due to the fact that the death of the bone is the last of a series of changes, of which the earlier consist of chronic inflammation, with hypertrophy and sclerosis.

4. That the symptoms of extensive necrosis, occurring in the course of chronic osteitis, in adults, may be expected to pursue, and do pursue a course which differs, in many respects, from that which is seen in the more common examples of extensive necrosis in the young.

5. That suppuration is not an early event, usually, in cases of necrosis from chronic osteitis.

6. That in such cases of necrosis, the endosteum as well as the periosteum contributes a large quantity of new bone.

7. That it may be well, for distinction's sake, to term this variety of necrosis, in which the sequestrum is enclosed within both periosteal and endosteal new bone, *intra-osseous*.

8. That there exist cases of intra-osseous necrosis, in which complete removal of the dead bone by surgical operation is, from the nature of its connections, a practical impossibility, and for which, therefore, if the sym-

ptoms are sufficiently distressing, amputation is the best remedy.

9. That, in favorable cases, when the disease is not extensive, the surrounding parts may heal, although some dead bone is permanently confined within its new sheath; the separation of the living from the dead being indefinitely postponed.

10. That the peculiarity of the case which forms the text of this paper is to be found rather in the rarity of spontaneous fracture, and of opportunities of examining the bone, in cases of necrosis from chronic osteitis, at an early stage, than in the nature of the case itself.

I have appended two drawings of typical examples of chronic osteitis with hypertrophy and sclerosis, in order that the conditions may be compared with those of the specimens of necrosis. In the *first* (Plate XI, fig. 3), it will be seen that the modelling of the bone is not quite completed, and the line of the original shaft with its periosteal and endosteal outgrowths is still capable of being traced.

At a later stage all distinction between the new and the old bone is lost; and at the same time, of course, all trace of the manner in which the hypertrophy has been effected is lost also (Plate XI, fig. 4).

DESCRIPTION OF PLATES X AND XI.

Necrosis without Suppuration.

PLATE X.

Fig. 1. Section of a femur removed by amputation at the hip-joint. Nearly the whole of the shaft has perished, and before the operation the bone had undergone so-called spontaneous fracture; but at no time, either before or after the operation, could any trace of suppuration be discovered.

Fig. 2. Section of a femur of which nearly the whole shaft has perished. There are several cloacæ leading, through the new bone laid down by the periosteum, into the narrow space by which it is separated from the sequestrum. Where the femur has not perished, it is hypertrophied and sclerosed.

PLATE XI.

Fig. 1. Section of a femur, of which a large portion of the shaft has perished. There is not in the periosteal new bone any cloaca leading into the narrow space around the sequestrum.

Fig. 2. Section of a tibia presumed to have belonged to the same person as the preceding specimen, but from the opposite limb. It is diseased in exactly the same manner, and there is no aperture leading through the new bone into the sequestrum-containing cavity.

Fig. 3. Section of a femur which has undergone hypertrophy and sclerosis, as the results of chronic inflammation. New bone has been laid down by both the periosteum and endosteum. The modelling of the various layers is not yet complete, the line of the original shaft being still plainly visible.

Fig. 4. Section of a tibia also affected by chronic inflammatory hypertrophy and sclerosis; but the modelling process is complete and all trace of the line of the original shaft is lost.

Fig. 1.



Fig. 2.





Fig. 1.

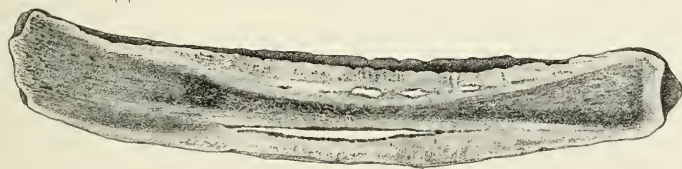


Fig. 2.

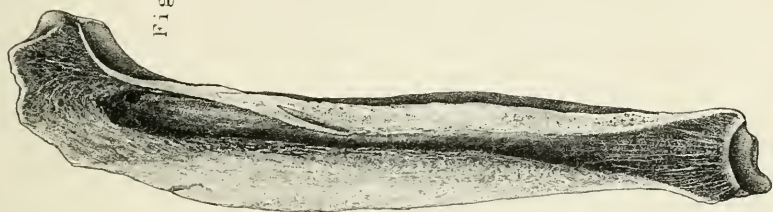


Fig. 4.

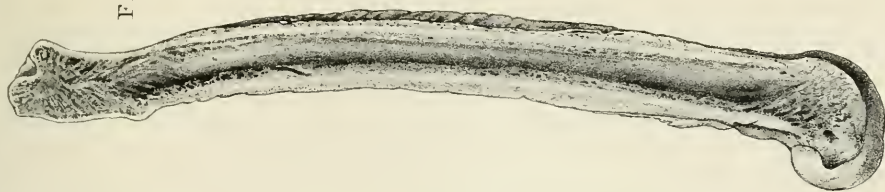


Fig. 3.

THREE HUNDRED
ADDITIONAL CASES OF OVARIOTOMY,
WITH REMARKS ON
DRAINAGE OF THE PERITONEAL CAVITY.

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(Received February 5th—Read February 27th, 1877.)

IN successive volumes of the 'Transactions' of this Society, between 1859 and 1872, some account has been published of 500 cases in which I had completed the operation of ovariectomy. I have now arranged in the same form as in the tables of former papers particulars of 300 additional cases, representing the whole of my practice of ovariectomy from the fifth to the eight hundredth operation.

In all the cases in this table, where the patient is stated to have been in hospital, the operations were performed in the Samaritan Hospital. This building has been enlarged by the addition of an upper story since the former series of cases was completed, and some possible sources of injury to operative cases have been removed, by treating many forms of uterine and other diseases in a branch

institution in another street. All these operations have been witnessed by medical visitors of this and other countries, and further details of any of the cases may be found in the hospital books by any one specially interested in the subject.

Where the name of the medical attendant is printed in Roman type the patient was operated on in a private house, free from the influence of any other patient. Where *italics* are used for the name of the medical attendant the operation was performed in one of the houses, now becoming more common in London, where, under the name of "Nursing Institution," or "Home for Invalids," or some such title, it is intended that a patient shall obtain the conjoint advantages of a hospital and of home or private apartments. There can be no doubt of the advantages of such houses to patients, or of the great convenience to the surgeon, provided the management is good. But they must always be open to the objection of subjecting one patient, more or less, to the influence of others in adjoining rooms or in the same house. And it is interesting to notice that, whereas in this series of 300 cases the mortality in private houses was 25·38 per cent., and in the Samaritan Hospital 25·60 per cent., it was 26·66 per cent. in the nursing homes. The numbers are as follows :

| | Cases. | Recoveries. | Deaths. | Mortality per cent. |
|------------------------|--------|-------------|---------|---------------------|
| Private houses . . . | 130 | 97 | 33 | 25·38 |
| Samaritan Hospital . . | 125 | 93 | 32 | 25·60 |
| Nursing homes . . . | 45 | 33 | 12 | 26·66 |

I may add that these houses are situated in Upper Wimpole Street, Great Marylebone Street, Manchester Street, Marylebone Road, and Blandford Square, positions not very different from that of the Samaritan Hospital. I am convinced that some of the deaths, both in hospital and in the nursing establishments, have been due to the injurious influence of other patients upon the subject of the operation; an influence which would not have been

felt in a private house. My belief is that, in the one case, if any important peritonitis follow the operation, the inflammation is almost always local, not attended by much effusion of serum, nor by elevation of temperature or other signs of fever or blood poisoning; whereas, under unfavorable sanitary conditions, the inflammation is diffused, is accompanied by the rapid effusion of a considerable amount of fluid, with great elevation of temperature and other indications of septicæmia. I am becoming more and more doubtful if we ever see this latter chain of symptoms, either in hospital or in healthy houses, if the patients are kept quite free from the access, by contagion or infection, of the poisonous material—solid, liquid, or gaseous—which acts as certainly as an inoculated particle of smallpox or vaccine virus, or as the inspiration of an infective atmosphere in scarlatina, and from which the patient is absolutely safe in the absence of the poison.

The average mortality in the 300 cases has been 25·66 per cent., 77 patients having died, and 223 recovered. In the sixth series of 100 cases there were 29 deaths, and in the seventh and eighth series 24 deaths in each. This result, so nearly that of the former series of 500 cases grouped together, has not fulfilled the hopes I ventured to express of diminishing mortality with increasing experience. But I think this may be explained by three reasons:—First, in cases of single cysts, I have become more disposed to advise tapping, and more hopeful that tapping may prove to be a radical cure when the cyst is really single, and have, therefore, performed ovariectomy less frequently than before in simple uncomplicated cases in young healthy women. Secondly, the proportion of severe or unfavorable cases has been increased by the fact that, as ovariectomy has become more generally practised, many of my friends have operated themselves in cases apparently favorable; whereas, when the result was more doubtful they have transferred their patients to my care. Thirdly, in several cases where formerly I should have thought it right to give so unfavorable a prognosis

to a patient and her friends or medical advisers, that they probably did not desire or approve of an operation, I have latterly been encouraged, by recoveries in some cases apparently almost hopeless, to give a more favorable prognosis, even under very unpromising local and general conditions. And although in some cases very unexpected recoveries have justified the operation, yet, in many other cases, the event has been what was feared, and the influence statistically upon the general mortality is quite appreciable.

On considering whether any recent modifications in operative procedure or after-treatment have influenced the results, the only subject I shall now submit for consideration is drainage of the peritoneal cavity.

In the early days of ovariectomy, when the pedicle was tied, was left within the abdominal cavity, and the ends of the ligature were brought out through some unclosed part of the incision, these threads acted as drains; fluid could escape and air could enter along their track until they could be withdrawn. When this method fell into disuse, and the end of the pedicle was secured outside, with the opening in the abdominal wall accurately closed around it; and also when the pedicle was secured by ligature or cautery, was left within, and the wound was completely closed, the improving results were attributed to the preservation of the peritoneum from the irritating effects of the ligature, which acted as a seton, set up suppurative changes, and permitted the entrance of air. Then, when instead of a mortality of 70 or 80 per cent., we could rejoice in a reduction to 20 or 30 per cent., and were hopeful of still better results, the cause of death in every case was carefully scrutinized, and it was felt that, as scarcely any of the deaths were due to shock or collapse or to primary hæmorrhage, and very few to acute peritonitis, nearly all were accounted for by a process of blood-poisoning. In the separation of adhesions some amount of blood which could not be entirely sponged away must be left in the peritoneal cavity, some ovarian

fluid, or some fluid effused by the peritoneum, would mix with the blood, degenerative changes, or decomposition, or putrefaction of this bloody fluid would take place, further secretion would be excited, and absorption of some portion of the poisonous mixture would be followed by deadly fever. Septicæmia, or septic peritonitis, then became recognised as the chief cause of death after ovariotomy, and cases were observed where the free escape of fetid gas and fluid, or their removal by repeated injections or washing out of the peritoneal cavity, were followed by recovery. All this was becoming well known, when it was advanced in favour of the practice not only of draining in every case where we had reason to fear that the patient was being injured by poisonous fluid retained within the peritoneal cavity, but for draining in every case of ovariotomy, making the drain an essential part of the operation, by passing a tube through the lowest part of the recto-vaginal pouch into the vagina before closing the abdominal wound, and either fastening it there as a simple vaginal drain, or bringing one end of the drainage-tube up through the partially closed opening in the abdominal wall, and leaving the other end passing through the vagina, in order to serve both as a constant drain and as a means of injecting water or antiseptic fluids at shorter or longer intervals.

The published cases in which this treatment had been adopted, and on the strength of which surgeons were invited to follow the practice, served to my mind rather as warnings than as examples; and, although in several instances very satisfactory cases were recorded to illustrate the practice. I took advantage of the opportunities afforded me in hospital practice to demonstrate that the passage of a tube into the vagina from the peritoneum, or in the opposite direction, was really a serious addition to the other necessary steps of the operation. I also felt that the tubes would be very likely to act as setons, and become the irritating cause of the very secretion which they were intended to remove, a secretion which was only observed

in a small proportion of cases where no drainage was provided for, and which could be removed by puncture in the few cases where it did form some hours or days after operation. It was not long before cases were published in which it had been found difficult to maintain the tubes in position, or to keep their channels clear, and where (notwithstanding free drainage of Douglas's pouch) the patients had died of septic peritonitis, and considerable quantities of bloody serum, with lymph or clots, had been found in the upper parts of the peritoneal cavity on either side of the vertebral column, quite unaffected by the vaginal drainage, and very imperfectly by the drain through the abdominal wall.

On examination of the notes of the 300 operations now brought before the Society, I find eight cases in which I made provision for drainage at the time of operation. In five of these cases I secured a glass tube (the lower end of which was passed down behind the uterus to the bottom of Douglas's pouch) between some part of the united edges of the opening in the abdominal wall. In two cases I made use of ligatures on the pedicle and on adhesions as drains, and in one case I passed a double india-rubber tube from Douglas's space through the vagina. In some of these cases I doubt whether either good or harm was done by the drainage; but in three or four of the cases where a glass tube was used I have no doubt it assisted in securing success, if it was not actually the means of saving life.

In eleven other cases, no provision for drainage having been made at the time of operation, fluid collected sooner or later afterwards, and in seven escaped either beside the clamp or between two of the stitches. In one case reddish serum, and in one purulent fluid, were removed by the insertion of a glass tube some days after operation. In two cases india-rubber tubes were used; in one through the abdominal wound, and in the other by vaginal puncture. Probably in some, if not in all, of these cases, drainage from the first would have done

good, by anticipating the collection of fluid and the symptoms caused by its decomposition and absorption, and by obviating the necessity for the subsequent drainage.

But as in only 19 cases out of 300 was primary or secondary drainage, to the best of my judgment and belief, called for, I think it may fairly be assumed that in a very large proportion of cases drainage at the time of the operation is unnecessary, and as it must necessarily prolong the anæsthesia and the exposure of the viscera, as well as complicate the process of repair, drainage should be reserved for those cases only where there is a great probability of blood, serum, or pus collecting in such quantity as to require removal.

Of the different modes of draining, that by the ends of ligatures from the pedicle or adhesions is the least satisfactory. The drainage is insufficient, some of the putrefying matter is retained, and the channel for the use of a syringe is incomplete and untrustworthy. India-rubber drainage-tubes are better than the ends of ligatures; but I greatly prefer strong glass tubes, and whenever drainage is thought necessary I would recommend that a glass tube should be passed down behind the uterus to the bottom of the recto-uterine pouch, and be fastened beside the pedicle if the clamp is used, or between two of the stitches if the pedicle is treated on one of the *intra-peritoneal* methods. A sponge—wet with a solution of carbolic or sulphurous acid or of chlorine or iodine—covers the end of the tube, and the dressing and bandage are then applied in the ordinary manner. When the sponge is saturated by the fluid which escapes from the tube, another sponge, charged with the antiseptic solution, should be substituted. To prevent clogging of the tube by blood-clot or lymph, a syringe, armed with an elastic tube, should be used for suction or antiseptic injections. So long as fluid escapes from the tube, or is drawn up by the syringe, the tube should be left, but it may be removed as soon as the discharge ceases. If left too

long, it may lead to the effusion of lymph, and the adhesion of neighbouring coils of intestine around it.

The combination of drainage with antiseptic injections and dressings must lead to further inquiry into the possibility of more certainly preventing septicæmia, by a more complete adoption of antiseptic precautions before, during, and after ovariectomy. And I hope to be able to bring this important question before the Society in another communication.

In the following Table, where the name of the Medical Attendant is in Roman type, the operation was performed in a private house ; where Italics are used, the operation was done in a Nursing Institution ; where Hospital is printed in Roman type, the patient was in the Samaritan Hospital.

TABLE OF 300 ADDITIONAL CASES OF COMPLETED OVARIOTOMY.

| No. | Medical attendant. | Date of operation. | Age. | Condition. | Adhesions. | Treatment of pedicle. | Weight of tumour. | Length of incision. | Result. | Subsequent History or Cause of Death. |
|-----|----------------------------|--------------------|------|------------|-------------------------------|-----------------------|-------------------|---------------------|----------------|--|
| 501 | Hospital | 1872 July | 21 | Single | None | Clamp and ligature | Pounds 27 | Inches 5 | Recovered | Well in 1876. |
| 502 | Dr. Busch, Ramsbeck..... | July | 51 | Married | Parietal | Clamp and ligature | 21 | 6 | Recovered | Well in 1876. |
| 503 | Hospital | July | 45 | Married | None | Clamp | 8 | 5 | Recovered | Well in July, 1873. |
| 504 | Dr. Ormerod, Brighton .. | July | 43 | Single | None | Clamp | 13 | 4 | Recovered | Well in 1876. |
| 505 | Hospital | July | 50 | Single | Pelvic | Ligature | ... | 7 | Died, 7th day | Pyæmic fever. |
| 506 | Dr. Kesteven | July | 56 | Married | Pelvic | Clamp | 12 | 6 | Died, 40 hours | Peritonitis. |
| 507 | Hospital | Aug. | 32 | Married | Omental. Pregnant... | Ligature | 26 | 6 | Recovered | Had 7 months' child day after operation. |
| 508 | Dr. Prior, Bedford..... | Aug. | 31 | Married | Omental and intestinal | Clamp | 18 | 4 | Recovered | Boys born Dec., 1873, and March, 1876. |
| 509 | Mr. W. Stewart | Aug. | 43 | Married | Parietal | Clamp | 8 | 5 | Recovered | Well in 1876. |
| 510 | Mr. Hall, Sheffield | Aug. | 42 | Married | Parietal and omental | Ligature | 21 | 5 | Recovered | Well Aug., 1876. |
| 511 | Dr. Williamson | Aug. | 54 | Married | Pelvic, omental, and parietal | Ligature | 52 | 5 | Died, 6th day | Peritonitis. |
| 512 | Dr. Fripp, Clifton | Aug. | 46 | Married | None | Ligature | 6 | 5 | Recovered | Well in 1876. |
| 513 | Dr. Pagenkopf, Moscow... | Aug. | 27 | Married | Parietal | Clamp and ligature. | 37 | 5 | Recovered | Well in 1876. |
| 514 | Dr. Docker, Boulogne..... | Aug. | 20 | Single | None | Both ovaries | 24 | 5 | Recovered | Well in 1876. |
| 515 | Mr. Mercer, Deal | Oct. | 31 | Single | None | Clamp | 21 | 5 | Died, 5th day | Septicæmia. |
| 516 | Dr. T. K. Chambers | Oct. | 37 | Married | Uterine | Ligature | 13 | 5 | Died, 42 hours | Septicæmia. |
| 517 | Dr. Churchill, Dublin..... | Oct. | 27 | Single | None | Clamp | 10 | 4 | Recovered | Well in 1876. |
| 518 | Dr. Thursfield, Leamington | Oct. | 60 | Single | None | Clamp | 20 | 4 | Recovered | Well in 1876. |

| No. | Medical attendant. | Date of operation. | Age. | Condition. | Adhesions. | Treatment of pedicle. | Weight of tumour. | Length of incision. | Result. | Subsequent History or Cause of Death. |
|-----|------------------------------|--------------------|------|------------|-----------------------------------|-----------------------|-------------------|---------------------|----------------|---|
| 519 | Dr. C. E. Roberts, Southgate | 1872 Oct. | 39 | Single | None. | Burst cyst ... | Pounds 36 | Inches 6 | Recovered | Died Sept., 1873. Cancer. |
| 520 | Dr. Ormerod, Brighton | Oct. | 67 | Married | Parietal and omental | Ligature | 6 | 5 | Died, 5th day | Peritonitis. |
| 521 | Hospital | Oct. | 28 | Married | Omental and parietal | Clamp | 21 | 5 | Recovered | Had twins in Sept., 1873. |
| 522 | Dr. Roche, Chelmsford | Nov. | 64 | Married | Parietal and omental | Clamp | 22 | 5 | Died, 13 hours | Exhaustion. |
| 523 | Dr. H'are | Nov. | 35 | Married | Omental and parietal | Clamp | 41 | 5 | Recovered | Two children since. |
| 524 | Mr. Reid, Canterbury | Nov. | 50 | Married | Omental, intestinal, and parietal | Clamp | 10 | 5 | Recovered | Well in Dec., 1876. |
| 525 | Hospital | Nov. 1873 | 25 | Single | None | Clamp | 25 | 5 | Died, 3rd day | Obstruction of intestine. |
| 526 | Hospital | Jan. | 58 | Married | Omental | Clamp | 11 | 5 | Died, 4th day | Peritonitis. |
| 527 | Dr. Sealy, Barbadoes | Jan. | 42 | Married | None | Clamp | 9 | 5 | Died, 7th day | Peritonitis. |
| 528 | Hospital | Jan. | 37 | Married | Parietal | Ligature | 19 | 5 | Died, 2nd day | Septicæmia. |
| 529 | Dr. Hawkesley | Jan. | 49 | Married | None | Clamp | 8 | 4 | Died, 4th day | Uremia from suppression of urine. |
| 530 | Hospital | Jan. | 22 | Single | Omental and parietal | Clamp | 28 | 4 | Recovered | Well a year after. |
| 531 | Hospital | Feb. | 56 | Married | Omental | Ligature | 17 | 5 | Recovered | Well Dec., 1876. |
| 532 | Hospital | Feb. | 32 | Married | None | Clamp | 13 | 4 | Recovered | Well in 1876. |
| 533 | Mr. Edgar Barker | Feb. | 44 | Married | Intestinal, omental, and parietal | No pedicle | 8 | 5 | Recovered | Well Dec., 1876. |
| 534 | Mr. Edgar Barker | Feb. | 42 | Married | None | Clamp | 23 | 4 | Recovered | Well in Dec., 1876. |
| 535 | Hospital | Feb. | 30 | Married | Omental | No pedicle | 33 | 5 | Recovered | Died of pleurisy one year after. |
| 536 | Dr. Churehill, Dublin | Feb. | 40 | Single | Omental | Ligature | 4 | 4 | Recovered | Well in 1876. |
| 537 | Dr. Oldham | Feb. | 57 | Married | Uterine | Pin and écraseur | 22 | 5 | Recovered | Well in 1876. |
| 538 | Hospital | Feb. | 31 | Single | None | Clamp | 23 | 5 | Recovered | Well in Jan., 1874. |
| 539 | Mr. Bishop, Tunbridge | Feb. | 31 | Single | Omental and parietal | Clamp | 11 | 4 | Recovered | Recovered after removal of other ovary. |

| | | | | | | | | | | | |
|-----|-----------------------------|-------|-----|---------|----------------------------------|-------|-----------------------|-----|---|----------------|--|
| 540 | Mr. Crompton, Birmingham | March | 31 | Single | None | | Clamp | 13 | 4 | Recovered | Well in 1876. |
| 541 | Hospital | March | 30 | Single | None | | Clamp | 19 | 4 | Died, 8th day | Septicæmia. |
| 542 | Dr. Watt, Black | March | 60 | Single | Parietal | | Clamp | 23 | 5 | Recovered | Well in 1876. |
| 543 | Hospital | March | 54 | Single | None | | Ligature | 12 | 5 | Died, 3rd day | Septicæmia. |
| 544 | Dr. Sharpe, Wootrich | March | 38 | Married | None | | Clamp | 17 | 4 | Recovered | Boy born Feb., 1876. |
| 545 | Dr. Rutherford, Palsborough | April | ... | Married | None | | Clamp | 16 | 4 | Recovered | Died of cancer 1874. |
| 546 | Dr. A. Brown, Islington | April | 33 | Single | None | | Clamp | 17 | 4 | Recovered | Well in 1876. |
| 547 | Dr. Evans, Hertford | April | 30 | Married | None | | Clamp | 16 | 4 | Recovered | Well Dec., 1876. |
| 548 | Hospital | April | 49 | Married | None | | Clamp | 19 | 4 | Died, 42 hours | Exhaustion. |
| 549 | Hospital | April | 29 | Married | Parietal | | Clamp | 30 | 6 | Recovered | Well in Jan., 1874. |
| 550 | Hospital | April | 50 | Married | Parietal | | Clamp | 50 | 6 | Recovered | Well in Jan., 1874. |
| 551 | Hospital | April | 20 | Single | Parietal | | Clamp | 20 | 4 | Recovered | Recovered after removal of second ovary in 1876. |
| 552 | Mr. Curtis, Alton | May | 42 | Single | None | | Ligature | 7 | 4 | Died, 3rd day | Peritonitis. |
| 553 | Mr. Raddock | May | 17 | Single | None | | Clamp | 18 | 4 | Died, 3rd day | Peritonitis. |
| 554 | Dr. Freund, Breslau | May | 36 | Single | None | | Clamp | 15 | 4 | Died, 48 hours | Septicæmia. |
| 555 | Hospital | May | 26 | Single | None | | Clamp | 13 | 5 | Died, 12th day | Septicæmia. |
| 556 | Mr. Hughes, Bromley | May | 43 | Single | Parietal | | Clamp | 14 | 4 | Recovered | Well in 1876. |
| 557 | Dr. Prince | May | 30 | Married | None | | Ligature. | 14 | 5 | Recovered | Died April, 1874. |
| 558 | Dr. Swayne, Clifton | June | 30 | Single | None | | Both ovaries Clamp | ... | 4 | Recovered | Cancer. Married in 1875. |
| 559 | Mr. Scattergood, Leeds | June | 41 | Married | Parietal | | Clamp | 38 | 5 | Died, 15th day | Cardiac embolism. |
| 560 | Hospital | June | 30 | Married | None | | Clamp | 18 | 5 | Recovered | Well in 1875. |
| 561 | Dr. Pagenkopff, Moscow | June | 42 | Married | Parietal | | Clamp | 17 | 6 | Recovered | |
| 562 | Hospital | June | 21 | Married | Parietal | | Clamp | 22 | 5 | Recovered | |
| 563 | Hospital | June | 58 | Married | None | | Clamp | 19 | 5 | Recovered | |
| 564 | Dr. Gonzalez, Riode Janeiro | June | 53 | Single | Parietal, intestinal, and pelvic | | Clamp | 125 | 6 | Recovered | Returned to Brazil. |
| 565 | Hospital | July | 28 | Single | None | | Clamp | 24 | 5 | Died, 5th day | Septic peritonitis. |
| 566 | Hospital | July | 21 | Married | Omental | | Clamp | 14 | 5 | Recovered | Two children born since. |
| 567 | Hospital | July | 51 | Single | Parietal | | Clamp | 26 | 5 | Recovered | Well in Feb., 1874. |
| 568 | Mr. Garraway, Faversham | July | 32 | Married | Parietal and omental | | Clamp | 21 | 5 | Recovered | Boy stillborn, 1875. Girl born, 1876. |

| No. | Medical attendant. | Date of operation. | Age. | Condition. | Adhesions. | Treatment of pedicle. | Weight of tumour. | Length of incision. | Result. | Subsequent History or Cause of Death. |
|-----|-----------------------------------|--------------------|------|------------|------------------------|-------------------------|-------------------|---------------------|----------------|---------------------------------------|
| 569 | <i>Dr. Thomas, Omagh</i> | 1873 | | | | | Pounds | Inches | | |
| 570 | Hospital | July | 34 | Married | None | Ligature | 10 | 5 | Recovered | Child born Aug., 1874. |
| | Hospital | July | 20 | Married | None | Ligature | 34 | 5 | Recovered | Well in 1876. |
| 571 | Hospital | July | 51 | Married | None | Clamp | 27 | 5 | Recovered | Well in 1876. |
| 572 | Hospital | July | 37 | Single | None | Ligature | 26 | 5 | Recovered | Well Dec., 1876. |
| 573 | <i>Dr. Corner</i> | Aug. | 20 | Single | None | Clamp | 17 | 5 | Recovered | Peritonitis. |
| 574 | <i>Dr. Guinness, Oxford</i> | Aug. | 29 | Married | Parietal and omental | Clamp | 9 | 6 | Died, 49 hours | Well Dec., 1876. |
| 575 | Dr. Bell | Aug. | 52 | Married | None | Clamp | ... | 5 | Recovered | Well in 1876. |
| 576 | Dr. F. E. Image, Bury St. Edmunds | Oct. | 40 | Married | Parietal | Ligature | 40 | 5 | Recovered | Well Dec., 1876. |
| 577 | Hospital | Oct. | 47 | Married | None | Clamp | 33 | 5 | Recovered | Well Dec., 1876. |
| 578 | Dr. Braxton Hicks | Oct. | 39 | Married | Omental | Pin and ligature | 16 | 6 | Died, 32 hours | Exhaustion. |
| 579 | Hospital | Oct. | 58 | Married | Omental | Clamp | 13 | 5 | Recovered | Well in 1876. |
| 580 | Dr. Chessall, Horley | Oct. | 32 | Single | Pelvic | Clamp | 13 | 5 | Recovered | Well in 1876. |
| 581 | Hospital | Oct. | 52 | Single | Intestinal | Clamp | 50 | 5 | Died, 28 hours | Exhaustion. |
| 582 | Dr. Brodie | Oct. | 22 | Married | Parietal | Clamp | ... | 5 | Recovered | Well in Dec., 1876. |
| 583 | Hospital | Oct. | 55 | Married | Parietal and omental | Ligature | 18 | 5 | Recovered | Well Dec., 1876. |
| 584 | Mr. Marriott, Swaffham | Oct. | 21 | Single | None | Sewed to abdominal wall | 18 | 5 | Died, 24th day | Pyæmic fever. |
| 585 | Mr. Riggall | Nov. | 47 | Married | None | Clamp | 12 | 5 | Recovered | Well in 1876. |
| 586 | Hospital | Nov. | 35 | Married | Omental and intestinal | Clamp | 15 | 5 | Recovered | Two children born since. |
| 587 | Hospital | Nov. | 55 | Single | None | Clamp | 12 | 5 | Recovered | Well in 1876. |
| 588 | <i>Dr. Hewer</i> | Nov. | 44 | Married | None | Clamp | 22 | 5 | Recovered | Well in 1876. |
| 589 | Hospital | Nov. | 34 | Married | Omental and parietal | Clamp | 21 | 6 | Recovered | Septicæmia. |
| 590 | <i>Dr. Giles, Oxford</i> | Dec. | 45 | Single | None | Clamp | 20 | 6 | Died, 8th day | Well in 1876. |
| 591 | Dr. Swayne, Clifton | Dec. | 44 | Single | Parietal | Clamp | 19 | 4 | Recovered | Well in 1876. |
| 592 | Hospital | Dec. | 29 | Married | Omental | Clamp | 22 | 5 | Died, 7th day | Septicæmia. |
| 593 | Hospital | Dec. | 24 | Single | None | Clamp | 15 | 5 | Recovered | Well Dec., 1876. |
| 594 | <i>Dr. Gage Brown</i> | Dec. | 41 | Single | Parietal | Clamp | ... | 4 | Recovered | Well in 1876. |

| | | | | | | | | | |
|---------------------------------------|-----------|----|---------|-----------------------------------|----------|-----|-----|----------------|--|
| 595 Hospital | Dec. | 43 | Married | Parietal and omental | Clamp | 21 | 5 | Recovered | Well in 1876. |
| 596 Hospital | Dec. | 46 | Married | None | Clamp | 32 | 5 | Recovered | Well Dec., 1876. |
| 597 Mr. Humby | Dec. 1874 | 56 | Married | Parietal | Ligature | 24 | 6 | Died, 21 hours | Exhaustion. |
| 598 Dr. Leslie, Alton | Jan. | 55 | Married | Parietal | Clamp | 21 | 5 | Died, 53 hours | Exhaustion. |
| 599 Hospital | Jan. | 24 | Single | None | Clamp | 35 | 5 | Recovered | Well in 1876. |
| 600 Mr. Winter, Brighton | Jan. | 50 | Single | Parietal | Clamp | 46 | 5 | Recovered | Well in 1876. |
| 601 Mr. Nunn, Colchester | Jan. | 32 | Single | None | Clamp | 12 | 4 | Recovered | Well in 1876. |
| 602 Hospital | Jan. | 33 | Married | Omental | Clamp | 24 | 5 | Recovered | Well in Dec., 1876. |
| 603 Hospital | Jan. | 22 | Single | None | Ligature | 16 | 4 | Recovered | Well Dec., 1876. |
| 604 Dr. Lane, San Francisco | Jan. | 8 | Single | None | Ligature | 2 | 4 | Recovered | Well in 1876. |
| 605 Dr. Highmore, Bradford-on-Avon | Jan. | 39 | Single | None | Clamp | 17 | 5 | Recovered | Died. Cancer of pericardium. Aug., 1875. |
| 606 Hospital | Feb. | 30 | Married | Omental | Clamp | 24 | 6 | Recovered | |
| 607 Hospital | Feb. | 49 | Married | None | Clamp | 17 | 5 | Recovered | |
| 608 Professor Dohrn, Marburg, Prussia | Feb. | 33 | Married | None | Clamp | 16 | 4 | Recovered | Well in 1876. |
| 609 Dr. Clifton, Leicester | Feb. | 52 | Married | Parietal | Clamp | 15 | 5 | Recovered | Well Dec., 1876. |
| 610 Hospital | Feb. | 51 | Single | Omental | Clamp | 26 | 5 | Died, 4th day | Congestion of lungs. |
| 611 Dr. Wyman, Putney | Feb. | 25 | Single | Intestinal | Clamp | 20 | 4 | Recovered | Died of phthisis. |
| 612 Hospital | Feb. | 52 | Single | None | Clamp | 12 | 5 | Died, 17th day | Clot in cerebral sinuses. |
| 613 Mr. Pitcher, Boston | Feb. | 49 | Married | None | Clamp | 16 | 5 | Recovered | Pulmonary congestion. |
| 614 Dr. Neil Arnott | March | 63 | Married | None | Clamp | 20 | 5 | Died, 2nd day | |
| 615 Hospital | March | 48 | Married | Omental and parietal | Ligature | 14 | ... | Recovered | Well in 1875. |
| 616 Hospital | March | 53 | Married | Omental, parietal, and intestinal | Clamp | 21 | ... | Recovered | Well in 1876. |
| 617 Dr. Borland, Boston, U.S. | March | 20 | Single | None | Clamp | 8 | 4 | Recovered | Well in 1876. |
| 618 Hospital | March | 50 | Married | Parietal | Clamp | 28 | 5 | Recovered | Well Dec., 1876. |
| 619 Hospital | April | 47 | Married | Omental | Clamp | 26 | 5 | Recovered | |
| 620 Hospital | April | 24 | Single | Omental and intestinal | Clamp | 16 | 5 | Recovered | Boy born July, 1876. |
| 621 Mr. Barrett, Pewsey, Wilts | April | 53 | Married | Omental | Clamp | ... | 5 | Recovered | Well Dec., 1876. |
| 622 Dr. Monro, Barnard Castle | April | 44 | Married | Parietal | Clamp | 17 | 5 | Died, 9th day | Purulent peritonitis. |
| 623 Hospital | April | 45 | Widow | None | Clamp | 12 | 4 | Recovered | |
| 624 Hospital | April | 29 | Single | Parietal | Clamp | 8 | 4 | Recovered | Well in 1876. |
| 625 Dr. Thomson, Torquay | April | 32 | Married | None | Clamp | 10 | 5 | Recovered | Well in 1876. |

| No. | Medical attendant. | Date of operation. | Age. | Condition. | Adhesions. | Treatment of pedicle. | Weight of tumour. | Length of incision. | Result. | Subsequent History or Cause of Death. |
|-----|-------------------------------|--------------------|------|------------|------------------------------------|-----------------------|-------------------|---------------------|----------------|---------------------------------------|
| | | 1874 | | | | | Pounds | Inches | | |
| 626 | Mr. Woodward, Tooting..... | May | 35 | Married | Omental and parietal | Clamp | 47 | 6 | Died, 3rd day | Septic peritonitis. |
| 627 | Mr. Harper, Holbeach..... | May | 31 | Married | Parietal, omental, and vesical | Clamp | 15 | 6 | Recovered | Well Dec., 1876. |
| 628 | Hospital | May | 47 | Married | Omental and parietal | Clamp | 30 | 5 | Died, 5th day | Septic peritonitis. |
| 629 | Mr. Nicholson, Stratford..... | May | 20 | Single | None | Clamp | 8 | 4 | Died, 4th day | Septicæmia. |
| 630 | Hospital | May | 46 | Married | Intestinal and uterine | Clamp | 24 | 5 | Died, 5th day | Septic peritonitis. |
| 631 | Dr. Bright, Forest Hill | May | 62 | Married | None | Clamp | 12 | 5 | Recovered | Well in 1876. |
| 632 | Hospital | May | 52 | Married | Parietal | Clamp | 27 | 5 | Recovered | |
| 633 | Mr. Everett, Worcester | May | ... | Married | Both ovaries | Clamp and ligature | 10 | 6 | Recovered | Well in 1876. |
| 634 | Dr. Britton, Clifton | May | 47 | Married | Omental | Clamp | 32 | 6 | Died, 22 hours | Septicæmia. |
| 635 | Dr. Veit, Bonn..... | May | 27 | Married | None | Clamp | 12 | 5 | Recovered | Well in 1876. |
| 636 | Hospital | May | 30 | Single | None. Both ovaries | Clamp and ligature | 27 | 5 | Recovered | Well in 1876. |
| 637 | Hospital | June | 34 | Married | Parietal | Clamp | 28 | 5 | Died, 3rd day | Hæmorrhage and septicæmia. |
| 638 | Hospital | June | 34 | Married | None | Clamp | 21 | 5 | Recovered | Well in 1876. |
| 639 | Mr. Baker, Birmingham | June | 45 | Married | Pelvic and omental | Clamp | 18 | 5 | Died, 5th day | Peritonitis. |
| 640 | Dr. Veit, Bonn | June | 30 | Single | None | Clamp | 9 | 5 | Died, 11th day | Clot in pulmonary artery. |
| 641 | Hospital | June | 20 | Single | None | Clamp | 12 | 4 | Recovered | |
| 642 | Dr. Wyld..... | June | 54 | Married | None | Clamp | 15 | 5 | Recovered | Well in 1876. |
| 643 | Dr. Snayne, Clifton | June | 48 | Married | Parietal and pelvic. Both ovaries | Clamp and ligature | 16½ | 5 | Died, 50 hours | Septicæmia. |
| 644 | Hospital | June | 58 | Married | Omental | Ligature | 27 | 6 | Died, 32 hours | Septicæmia. |
| 645 | Hospital | June | 58 | Married | Omental and parietal. Both ovaries | Ligature (both) | 18 | 5 | Died, 5th day | Septic peritonitis. |
| 646 | Hospital | July | 26 | Married | Omental | Clamp | 16½ | 5 | Recovered | Child born 1876. |
| 647 | Dr. Winckel, Dresden..... | July | 24 | Married | Parietal and omental | Clamp | ... | 4 | Recovered | Well in 1876. |
| 648 | Dr. Gage Brown | July | 39 | Married | Omental | Clamp | 24 | 5 | Recovered | Child born Dec., 1875. |

| | | | | | | | | | | | |
|-----|-------------------------|------|-----|---------|---|--------------|--------------------|-----|-----|----------------|---------------------------------------|
| 649 | Mr. Hewer | July | 54 | Single | Parietal | | Clamp | 21 | ... | Recovered | Well in 1876. |
| 650 | Dr. Magrath, Teignmouth | July | 16 | Single | Parietal, omental, and intestinal | | Clamp | 10 | 6 | Recovered | Well Dec., 1876. |
| 651 | Dr. Roberts, Port Madoc | July | 62 | Married | None | | Clamp | 13 | 4 | Recovered | Peritonitis. |
| 652 | Hospital | July | 37 | Married | Intestinal, vesical, and uterine | | Clamp and ligature | 33 | 6 | Died, 26 hours | Well in 1876. |
| 653 | Mr. Hewlett, Harrow | July | 25 | Single | None. | Both ovaries | Ligature | 20 | 5 | Recovered | Well in 1876. |
| 654 | Hospital | Aug. | 24 | Single | None | | Clamp | 20 | 5 | Recovered | Well in 1876. |
| 655 | Mr. Burton, Blackheath | Aug. | 32 | Married | None | | Clamp | 38½ | 5 | Died, 50 hours | Septicæmia. |
| 656 | Dr. Horsford, Stratford | Oct. | 40 | Married | Parietal and omental | | Ligature | 14 | 5 | Recovered | Died in 1875 of cancer. |
| 657 | Hospital | Oct. | 25 | Single | None | | Clamp | 17½ | 5 | Recovered | Well in 1876. |
| 658 | Mr. Walker, Wakefield | Oct. | 41 | Widow | None | | Clamp | 11 | 5 | Recovered | Died Feb., 1875. |
| 659 | Dr. Owen Rees | Oct. | 54 | Married | Parietal and omental. | | Ligatures | 55 | 8 | Recovered | |
| 660 | Hospital | Nov. | 24 | Married | Both ovaries | | Clamp | 16 | 5 | Recovered | |
| 661 | Mrs. Garrett-Anderson | Nov. | ... | Single | Parietal, omental, vesical, and uterine | | Ligatures | 10 | 5 | Recovered | Well Oct., 1875. |
| 662 | Hospital | Nov. | 54 | Single | None. | Both ovaries | Clamp | 15 | 5 | Recovered | Well Dec., 1876. |
| 663 | Mr. Coates, Salisbury | Nov. | 54 | Married | Parietal and omental | | Clamp | 13 | ... | Recovered | Well in 1876. |
| 664 | Dr. Pauly, Ebersvalde | Nov. | 42 | Married | None | | Clamp and ligature | 12 | 5 | Recovered | Well in 1876. |
| 665 | Dr. Gordon, Belfast | Nov. | 27 | Married | Parietal | | Clamp | 9 | 5 | Recovered | Child born Oct. 1876 |
| 666 | Mr. Clover | Dec. | 24 | Single | Parietal | | Clamp | 15 | 5 | Recovered | Well in 1876. |
| 667 | Dr. Wood, New York | Dec. | 45 | Married | Uterine | | Ligature | 10 | 5 | Died, 13th day | Obstruction of intestine. |
| 668 | Mr. Taylor, Guildford | Dec. | 55 | Single | Omental and intestinal. | | Ligatures | 15 | 6 | Died, 30 hours | Peritonitis. |
| 669 | Hospital | Dec. | 35 | Single | Omental | Both ovaries | Clamp | 17 | 6 | Died, 4th day | Septicæmia. |
| 670 | Sir W. Gull, Bart. | Dec. | 16 | Single | None | | Clamp | 18 | 4 | Recovered | Well in 1876. |
| 671 | Hospital | Dec. | 21 | Single | Parietal and uterine | | Clamp | 41 | 5 | Recovered | Well in 1876. |
| 672 | Hospital | 1875 | 51 | Widow | Omental | | Clamp | 9 | 5 | Recovered | Well in 1876. |
| 673 | Hospital | Jan. | 15 | Single | None | | Clamp | 2 | 3 | Recovered | Died of broncho-pneumonia, May, 1875. |
| 674 | Mr. Payne, Cambridge | Jan. | 31 | Single | Parietal | | Clamp | 26 | 5 | Recovered | Well Dec., 1876. |

| No. | Medical attendant. | Date of operation. | Age. | Condition. | Adhesions. | Treatment of pedicle. | Weight of tumour. | Length of incision. | Result. | Subsequent History or Cause of Death. |
|-----|--------------------------------|--------------------|------|------------|----------------------------------|-----------------------|-------------------|---------------------|----------------|---------------------------------------|
| 675 | Dr. Wharton Hood..... | 1875 | ... | Married | Parietal & mesenteric | Clamp | Pounds 13 | Inches 5 | Recovered | Well in 1876. |
| 676 | Hospital | Jan. | 57 | Married | Parietal | Clamp | 4 | 5 | Recovered | Well in 1876. |
| 677 | Mr. Forster, Daventry | Jan. | 34 | Single | Omental | Ligature | 14 | 5 | Recovered | Well in 1876. |
| 678 | Hospital | Jan. | 44 | Single | Parietal, intestinal, and pelvic | Clamp | 4 | 4 | Died, 28 hours | Peritonitis. |
| 679 | Hospital | Jan. | 49 | Married | Parietal | Clamp | 13 | 5 | Died, 8th day | Septicæmia. |
| 680 | Hospital | Jan. | 38 | Married | Omental | Clamp | 30 | 5 | Recovered | |
| 681 | Dr. Fawcett, Cambridge | Feb. | 30 | Single | Omental | Clamp | 11 | 5 | Recovered | Well in 1876. |
| 682 | Hospital | Feb. | 47 | Single | Parietal | Clamp | 11 | 5 | Recovered | Well Dec., 1876. |
| 683 | Dr. Lanchester, Croydon | Feb. | 25 | Married | Parietal and omental | Clamp | ... | 4 | Recovered | Well in 1876. |
| 684 | Dr. Prell, Hamburg | Feb. | 39 | Married | None | Clamp | 10 | 4 | Recovered | Well in 1876. |
| 685 | Hospital | Feb. | 25 | Single | None. Both ovaries | Clamp | 22 | 5 | Recovered | Well Dec., 1876. |
| 686 | Hospital | Feb. | 16 | Single | None | Clamp | 14 | 5 | Recovered | Well Dec., 1876. |
| 687 | Dr. Weir, Malvern | Feb. | 64 | Married | No true pedicle | Clamp | 15 | 5 | Recovered | Well in 1876. |
| 688 | Hospital | Feb. | 32 | Married | Parietal and omental | Clamp | 9 | 5 | Recovered | Child born Aug., 1876. |
| 689 | Dr. Griffith, Camberwell | March | 36 | Single | None | Clamp | 20 | 5 | Died, 3rd day | Peritonitis. |
| 690 | Hospital | March | 30 | Single | Pelvic | Clamp | 22 | 5 | Recovered | Well Dec., 1876. |
| 691 | Hospital | March | 38 | Married | Parietal and omental | Ligature | 55 | 6 | Recovered | Child born July, 1876. |
| 692 | Dr. Pagenkopf, Moscow | March | 55 | Married | Intestinal, uterine, &c. | Clamp and ligature | 33 | 6 | Died | Obstruction of intestine. |
| 693 | Dr. Hill, Lymington | March | 60 | Single | None | Clamp | 14 | 5 | Recovered | Well in 1876. |
| 694 | Dr. Rice | April | ... | Married | Parietal and omental | Clamp | 22 | 5 | Recovered | Well in 1876. |
| 695 | Dr. Manifold, Liverpool | April | 56 | Married | Parietal and omental | Clamp | 13 | 5 | Recovered | Well in 1876. |
| 696 | Mr. Robinson, Bedford | April | ... | Married | None | Clamp | ... | 5 | Recovered | Stillborn boy April, 1876. |
| 697 | Mr. Shepherd, Worcester | April | 24 | Single | None | Clamp | 11 | 4 | Recovered | Well in 1876. |
| 698 | Dr. Goldschmidt, Hamburg | April | 39 | Married | None | Clamp | 10 | 4 | Recovered | Well in 1876. |
| 699 | Dr. Newman, Stamford | April | 63 | Married | Omental | Ligature | 15 | 6 | Recovered | Well Dec., 1876. |
| 700 | Hospital | April | 46 | Married | Omental | Clamp | 22 | 6 | Recovered | |

| 701 Hospital | April | 46 | Single | Parietal, omental, and between the two tumours | Ligatures (both pedicles) | 20 | 7 | Died, 26 hours | Exhaustion. |
|----------------------------------|-------|-----|---------|---|---------------------------|-----|---|----------------|--------------------------|
| 702 Dr. Holman, Reigate | May | 23 | Single | Parietal | Clamp | 14 | 4 | Died, 3rd day | Septicæmia. |
| 703 Dr. Kugler, Stettin | May | 34 | Married | None | Clamp | 77 | 5 | Recovered | Well in 1876. |
| 704 Mr. Doid | May | 27 | Married | None | Clamp | 9 | 4 | Recovered | Well in 1876. |
| 705 Mr. Barker | May | ... | Married | Parietal and omental | Clamp | ... | 5 | Recovered | Well in 1876. |
| 706 Mr. Orton, NARBOROUGH | May | ... | Married | None | Clamp | 10 | 5 | Recovered | Well in 1876. |
| 707 Hospital | May | 21 | Single | None | Clamp | 19 | 6 | Recovered | Well in 1876. |
| 708 Hospital | June | 46 | Married | Omental and intestinal | Clamp | 42 | 7 | Died, 10th day | Peritonitis. |
| 709 Mr. Blackstone | June | 47 | Widow | Omental | Clamp | 26 | 5 | Recovered | Well Dec., 1876. |
| 710 Dr. Synes Thompson | June | 18 | Single | None | Ligature | 4 | 6 | Died, 9th day | Peritonitis. |
| 711 Dr. Griffith, Swansea | June | 24 | Single | None | Ligature | ... | 5 | Recovered | Well in 1876. |
| 712 Mr. Copestake, Derby | June | 57 | Single | Parietal, omental, and intestinal. Both ovaries | Ligature (both) | 13 | 5 | Recovered | Died Oct., 1876. Cancer. |
| 713 Hospital | June | 70 | Widow | Intestinal | Clamp | 29 | 5 | Recovered | Well Dec., 1876. |
| 714 Hospital | June | 32 | Married | Omental | Ligature | 19 | 5 | Died, 6th day | Septicæmia. |
| 715 Dr. Rooke, Cheltenham | July | 33 | Married | Omental | Clamp | 21 | 5 | Recovered | Well in 1876. |
| 716 Mr. Turner, Hereford | July | 33 | Married | Omental and intestinal | Clamp | 11 | 5 | Recovered | Well in 1876. |
| 717 Hospital | July | 52 | Married | Omental | Clamp | 22 | 5 | Recovered | Well in 1876. |
| 718 Dr. Johnson, Tunbridge Wells | July | 60 | Single | Omental and intestinal | Clamp | 10 | 5 | Recovered | Well in 1876. |
| 719 Hospital | July | 46 | Widow | Omental | Clamp | 26 | 6 | Recovered | Well Dec., 1876. |
| 720 Dr. Dill, Brighton | July | 39 | Single | None | Clamp | 5 | 7 | Died, 6th day | Septicæmia. |
| 721 Hospital | Oct. | 29 | Married | Parietal and omental | Clamp | 12 | 5 | Recovered | Well in 1876. |
| 722 Hospital | Oct. | 31 | Married | Parietal and omental | Clamp | 33 | 5 | Recovered | Well and pregnant 1876. |
| 723 Hospital | Oct. | 45 | Single | Parietal, omental, uterine, and vesical | Clamp | 28 | 6 | Died, 3rd day | Septicæmia. |
| 724 Mr. F. Hutchinson | Oct. | 31 | Single | None | Clamp | 12 | 4 | Recovered | Well in 1876. |
| 725 Hospital | Oct. | 40 | Married | Omental | Clamp | 12 | 5 | Recovered | Well Nov., 1876. |
| 726 Hospital | Nov. | 25 | Single | None. Both ovaries | Clamp and ligature | 30 | 5 | Died, 2nd day | Septicæmia. |
| 727 Dr. Edis | Nov. | 51 | Married | Parietal and omental | Clamp | 20 | 5 | Recovered | Well Dec., 1876. |
| 728 Hospital | Nov. | 59 | Widow | Parietal and omental | Ligature | 15 | 5 | Recovered | Well June, 1876. |
| 729 Dr. Percy Boulton | Nov. | 24 | Single | Parietal, omental, intestinal, and uterine | Clamp | 25 | 5 | Died, 8th day | Exhaustion (?). |

| No. | Medical attendant. | Date of operation. | Age. | Condition. | Adhesions. | Treatment of pedicle. | Weight of tumour. | Length of incision. | Result. | Subsequent History or Cause of Death. |
|-----|--------------------------|--------------------|------|------------|-----------------------------------|-----------------------|-------------------|---------------------|----------------|---------------------------------------|
| | | 1875 | | | | | Pounds | Inches | | |
| 730 | Mr. J. W. Allen | Nov. | 54 | Widow | None | Clamp | 13 | 5 | Recovered | Well in 1876. |
| 731 | Hospital | Nov. | 28 | Single | Parietal and omental | Clamp | 29 | 5 | Died, 45 hours | Septic peritonitis. |
| 732 | Hospital | Nov. | 29 | Single | None | Clamp | 41 | 5 | Recovered | Well Nov., 1876. |
| 733 | Sir H. Thompson | Nov. | 21 | Single | None | Clamp | 11 | 4 | Recovered | Well in 1876. |
| 734 | Mr. Edgar Barker | Nov. | 38 | Widow | Intestinal | Clamp | 29 | 5 | Recovered | Well in 1876. |
| 735 | Mr. Foster, Huntingdon | Nov. | 39 | Single | Omental | Clamp | 10 | 4 | Recovered | Well in 1876. |
| 736 | Dr. Love, Lynn | Dec. | 25 | Single | None | Clamp | 6 | 4 | Recovered | Well Nov., 1876. |
| 737 | Dr. Scott, Huddersfield | Dec. | 35 | Single | None | Clamp | 14 | 4 | Died, 19th day | Intestinal obstruction. |
| | | 1876 | | | | | | | | |
| 738 | Mr. Morant Baker | Jan. | 28 | Single | None | Clamp | 15 | 5 | Died, 2nd day | Septicæmia. |
| 739 | Hospital | Jan. | 52 | Single | Omental | Clamp | 10 | 5 | Recovered | Well in 1876. |
| 740 | Dr. Smart, Hackney | Jan. | 45 | Married | Parietal, omental, and intestinal | Clamp | 17 | 6 | Recovered | Well in 1876. |
| 741 | Mr. Manifold, Liverpool | Jan. | 36 | Married | Parietal | Clamp | 11 | 5 | Recovered | Well Nov., 1876. |
| 742 | Hospital | Jan. | 27 | Married | None | Clamp | 9 | 4 | Recovered | Well Nov., 1876. |
| 743 | Mr. Proctor, Tunstall | Jan. | 35 | Married | None | Clamp | 47 | 5 | Recovered | Well Nov., 1876. |
| 744 | Hospital | Feb. | 27 | Single | Parietal | Clamp | 13 | 5 | Recovered | Well Nov., 1876. |
| 745 | Dr. Norton | Feb. | 26 | Married | Parietal and omental | Clamp | 15 | 5 | Recovered | Well Dec., 1876. |
| 746 | Dr. Bright, Forest Hill | Feb. | 23 | Single | Intestinal | Clamp | 9 | 5 | Recovered | Well Nov., 1876. |
| 747 | Dr. Herzfeld, Hamburg | Feb. | 54 | Single | Omental | Ligature | 15 | 5 | Died, 6th day | Septicæmia. |
| 748 | Hospital | Feb. | 32 | Married | Omental and pelvic | Clamp | 11 | 5 | Recovered | Well and pregnant Dec., 1876. |
| 749 | Dr. Nefel, New York | Feb. | 26 | Single | None | Clamp | 9 | 4 | Recovered | Well Nov., 1876. |
| 750 | Hospital | Feb. | 27 | Single | None | Clamp | 16 | 5 | Recovered | Well Dec., 1876. |
| 751 | Hospital | March | 48 | Married | Pelvic | Clamp | 8 | 5 | Recovered | Well Dec., 1876. |
| 752 | Dr. Kidd, Dublin | March | 36 | Married | Parietal and omental. | Ligature | ... | 5 | Died, 7th day | Exhaustion after delivery. |
| 753 | Hospital | March | 26 | Single | Pregnancy | Clamp | 11 | 5 | Recovered | Well Oct., 1876. |
| 754 | Dr. Frasch, Naugard | April | 42 | Single | Omental | Clamp | 31 | 6 | Died, 6th day | Peritonitis. |
| 755 | Dr. De Roubaix, Brussels | April | 32 | Married | Parietal, omental, and vesical | Ligatures. | 20 | 5 | Died, 8 weeks | Pelvic abscess. |
| | | | | | | Both ovaries | | | | |

| | | | | | | | | | | |
|----------------------------------|-------|----|---------|--|----------------------|---------------------|-----|---|---------------|---------------------|
| 756 Dr. Day | April | 27 | Married | None | Parietal and omental | Clamp | 10 | 5 | Recovered | Well Dec., 1876. |
| 757 Hospital | April | 39 | Married | Parietal and omental | Clamp | Ligatures. | 26 | 6 | Recovered | Well Nov., 1876. |
| 758 Dr. Kidd | April | .. | Single | Omental | Both ovaries | Clamp | 15 | 5 | Recovered | Well Dec., 1876. |
| 759 Mr. Whittington, Tuxford | April | 53 | Married | Parietal | .. | Clamp | ... | 5 | Recovered | Well Nov., 1876. |
| 760 Hospital | April | 26 | Single | Omental | .. | Clamp | 9 | 5 | Died, 7th day | Septic peritonitis. |
| 761 Hospital | April | 66 | Married | Parietal | .. | Clamp | 19 | 5 | Recovered | Well Dec., 1876. |
| 762 Mr. Harrison, Chester | April | 59 | Married | Omental & mesenteric | .. | Ligature | 25 | 5 | Recovered | Well Dec., 1876. |
| 763 Hospital | May | 47 | Single | None | .. | Clamp and ligature. | 19 | 5 | Recovered | Well Dec., 1876. |
| 764 Hospital | May | 21 | Single | None | .. | Both ovaries | 9 | 5 | Recovered | Died Oct., 1876. |
| 765 Hospital | May | 30 | Married | None | .. | Clamp | 18 | 5 | Recovered | Cancer. |
| 766 Dr. McClintock, Dublin | May | 18 | Single | None | .. | Clamp | 23 | 5 | Recovered | Well Nov., 1876. |
| 767 Hospital | May | 51 | Married | Parietal and omental | .. | Clamp | 23 | 5 | Recovered | Well Nov., 1876. |
| 768 Dr. Thomson, Armagh | May | 56 | Single | Parietal | .. | Clamp | 17 | 5 | Recovered | Well Nov., 1876. |
| 769 Hospital | May | 23 | Single | None | .. | Clamp | 2 | 4 | Recovered | Well Dec., 1876. |
| 770 Hospital | June | 34 | Single | Parietal and intestinal | .. | Clamp | 14 | 5 | Died, 5th day | Septic peritonitis. |
| 771 Hospital | June | 41 | Married | Omental | .. | Clamp | 30 | 5 | Recovered | Well Nov., 1876. |
| 772 Hospital | June | 29 | Single | Parietal | .. | Ligature | 14 | 5 | Recovered | Well Nov., 1876. |
| 773 Mr. Nason, Stratford-on-Avon | June | 54 | Single | None | .. | Ligature | 24 | 5 | Recovered | Well Nov., 1876. |
| 774 Dr. Priestley | June | 46 | Married | None | .. | Ligature. | 13 | 5 | Recovered | Well Nov., 1876. |
| 775 Hospital | June | 36 | Single | None | .. | Both ovaries | 16 | 5 | Recovered | Well Dec., 1876. |
| 776 Hospital | June | 29 | Single | None | .. | Clamp | 13 | 5 | Recovered | Well Dec., 1876. |
| 777 Hospital | July | 20 | Single | None | .. | Ligature | 10 | 5 | Recovered | Well Dec., 1876. |
| 778 Dr. Iliewicz, Jerusalem | July | 18 | Single | None | .. | Clamp | 19 | 5 | Recovered | Well Nov., 1876. |
| 779 Hospital | July | 27 | Single | Parietal, omental, uterine, and the two ovaries bound together | .. | Ligature | 5 | 5 | Died, 4th day | Septicæmia. |
| 780 Mr. Lowe, Burton-on-Trent | July | 48 | Married | Parietal and omental | .. | Clamp | 12 | 5 | Died, 7th day | Peritonitis. |
| 781 Hospital | July | 47 | Single | None | .. | Clamp | 24 | 4 | Recovered | Well Nov., 1876. |
| 782 Mr. Rigden, Lewis | July | 77 | Single | Parietal, omental, and vesical | .. | Clamp | 23 | 5 | Died, 6th day | Exhaustion. |

| No. | Medical attendant. | Date of operation. | Age. | Condition. | Adhesions. | Treatment of pedicle. | Weight of tumour. | Length of incision. | Result. | Subsequent History or Cause of Death. |
|-----|-----------------------------|--------------------|------|------------|-----------------------------------|-----------------------|-------------------|---------------------|----------------|---|
| 783 | Sir H. Thompson..... | 1876 | 52 | Single | Parietal and intestinal | Ligature | Pounds | Inches | | |
| 784 | Mr. Archer | July | 37 | Single | Parietal, hepatic, and intestinal | Clamp | 43 | 6 | Died, 10th day | Peritonitis. Well Dec., 1876. |
| 785 | Dr. Coates, Bath..... | July | 32 | Single | None | Clamp | 9 | 5 | Recovered | Well Nov., 1876. |
| 786 | Dr. Hawkesley..... | July | 27 | Single | None | Clamp | 7 | 5 | Recovered | Well Dec., 1876. |
| 787 | Hospital | July | 31 | Single | None | Clamp | 15 | 5 | Recovered | Well Nov., 1876. |
| 788 | Mr. Ceely, Aylesbury | July | ... | Single | None | Clamp and ligature. | 18 | 5 | Recovered | Well Dec., 1876. |
| 789 | Dr. Hodder, Toronto | Aug. | ... | Married | Intestinal, pelvic, and uterine | Both ovaries Ligature | 9 | 5 | Recovered | Well Nov., 1876. |
| 790 | Mr. Crosby, Salford..... | Aug. | 34 | Married | Omental | Ligature. | 7 | 5 | Recovered | Well Dec., 1876. |
| 791 | Sir William Gull, Bart. ... | Aug. | ... | Married | None | Both ovaries | 7 | 5 | Recovered | Well Dec., 1876. |
| 792 | Dr. Schonfeldt, Labes | Aug. | 38 | Single | None | Clamp | 10 | 4 | Recovered | Well Dec., 1876. |
| 793 | Mr. Hodgson, Brighton ... | Sept. | 51 | Married | Parietal and omental | Clamp and ligature | 12 | 5 | Recovered | Well Dec., 1876. |
| 794 | Dr. Clarke, Huddersfield... | Sept. | 52 | Married | Omental | Clamp | 12 | 5 | Died, 14th day | Peritonitis. |
| 795 | Hospital | Oct. | 54 | Single | Parietal | Clamp | 44 | 5 | Recovered | Well Dec., 1876. |
| 796 | Dr. Cardozo, Richmond ... | Oct. | 45 | Single | None | Ligature. | 16 | 5 | Recovered | Well Dec., 1876. |
| 797 | Hospital | Oct. | 29 | Single | Intestinal, vesical, and uterine | Both ovaries Ligature | 9 | 5 | Recovered | Well Dec., 1876. |
| 798 | Dr. Roberts | Oct. | 41 | Married | None. Pregnant..... | Clamp | 7 | 4 | Recovered | Pregnant and well Dec., 1876. |
| 799 | Hospital | Oct. | 26 | Single | Parietal | Clamp | 31 | 4 | Recovered | Well Dec., 1876. |
| 800 | Hospital | Oct. | 33 | Married | Intestinal and pelvic | Ligatures. | 19 | 4 | Died, 4 weeks | Peritonitis, and tubercular cavities in lung. |

THE PATHOLOGICAL ANATOMY
OF
CANINE "CHOREA."

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THE two cases which form the subject of this communication were examples of the disease of the dog which is known as "chorea," but which differs somewhat in its characters from the chorea of man. Its chief feature is the occurrence of quick contractions in isolated muscles or groups of muscles, the contractions being separated by considerable intervals, and resembling those in the affection which has been described as "chorea electrica." There is an absence of the spontaneous fidgetty movements and inco-ordination, which are so characteristic of chorea as it is usually seen in man.

In the two cases the movements were similar in character, but differed in extent and degree. In the slighter case they were limited to one fore leg; in the

other they were widely distributed, affecting all four limbs as well as the muscles of the head and neck. In each animal the effect of section of the cervical spinal cord was observed; in the former the movements persisted for a short time, in the latter they ceased at once. In the former only slight morbid changes were discovered in the nerve-centres; in the latter the cerebellum, spinal cord, and to a less extent the medulla oblongata, were the seat of extensive and unusual alterations.

It may be convenient to describe first the case in which the symptoms and morbid appearances were most marked.

CASE 1.—A retriever, six months old, which had lived chiefly in a dwelling house, was well until, at four months old, it had a mild attack of distemper. When it had nearly recovered from the distemper, slight twitching was observed in the left foreleg. This increased, and extended to the hind legs, which became affected at the same time, and nearly to the same extent. The right foreleg was then also attacked, and the twitching soon became more severe in it than in the hind legs. Soon all the muscles of the trunk and neck were involved, including the extrinsic laryngeal and the lingual muscles. Those of the eyeballs were free. Weakness in the limbs was then observed, noticed first in the hind limbs; the animal became unable to walk, and on an attempt to do so the body was curved, with the convexity to the right. These symptoms continued without abatement.

When it came under our observation, two months after the commencement of the affection, no change in temper was observed, and there was little evidence of pain. The animal was restless, frequently changing its posture. The muscular twitchings were then general; the contractions quick and sudden, like those caused by the interrupted voltaic current. The muscles of the same part contracted together, *e. g.*, those of the hind legs and tail, but the spasm of the hind legs and of the forelegs was not always simultaneous. The contractions occurred in the same muscles six or

seven times in a minute ; usually there were two or three in rapid succession, and then a long pause. They were more marked while the animal was at rest than when a movement was attempted. The weakness of the limbs, especially of the hind limbs, was very considerable. The dog could just stand, but on attempting to walk the hind legs gave way, and it fell. When lying it was able to move them freely. The tail could be moved with vigour. All the muscles of the right side seemed weaker than those of the left, and thus the curve of the trunk was produced. There was no obvious weakness of the muscles of the head or jaw.

Sensation was considerably diminished in the posterior half of the body. A pin could be driven in without causing the animal to cry or to wake from sleep. Pinching the tail was unfelt. Sensation in the fore legs seemed lessened, but not abolished. A prick caused the animal to look at the part, but not to withdraw the leg. Sensation seemed normal about the shoulders, the neck and the head. The animal had a habit of nibbling at the fore legs, as if they were the seat of some disordered sensation.

There was no affection of the organs of special sense. There was no paralysis of the sphincters.

The dog was placed under the influence of ether and the twitchings completely ceased. When it was allowed to recover partially from the influence of the ether the twitchings recommenced. The spinal cord was afterwards divided below the occiput, and artificial respiration performed by means of a tube previously introduced into the trachea. As soon as the dog had recovered from the effects of the ether, twitchings recommenced in the jaw and the muscles of the face and head, including the external laryngeal muscles, but there were no twitchings in any parts supplied from the spinal cord below the section during the four or five minutes which elapsed before the animal was killed. (The interpretation to be placed on this fact will be considered presently.)

A careful post-mortem examination revealed no disease of internal organs. No entozoa were found. The heart was healthy. The muscles to the naked eye and the microscope appeared perfectly healthy. The nerve centres were examined in a recent state and after hardening.

No lesion was discovered in the cerebral hemispheres or the central ganglia. On the other hand the spinal cord, the medulla oblongata, and the cerebellum presented extensive alteration.

Examination of the spinal cord in a fresh state (immediately after death) revealed little change. No areas of congestion or ecchymosis were visible. The white substance appeared normal in consistence and tint. Nerve-cells from the anterior cornua appeared somewhat swollen and unduly granular, but contained no pigment.

After hardening, however, extensive lesions were apparent. The most conspicuous change was the infiltration of limited areas with small round "lymphoid" cells, which varied in size from $\frac{1}{3000}$ to $\frac{1}{4000}$ inch, the average being $\frac{1}{3500}$ (.0075 mm.). In most of these cells no nucleus was visible, but in some a large round nucleus occupied about half or two thirds of the area of the cell. These cells were not uniformly infiltrated in the affected areas. In the white substance they were aggregated in tracts, often branching, and extending through part or the whole of the affected column (Pl. XII, figs. 1, 2, 3, and 4). When the infiltration affected the grey substance it was more uniform, but here also the corpuscles were aggregated in masses, as well as scattered. The aggregations in both grey and white matter corresponded to the course of vessels, or of the larger connective-tissue septa in which the vessels run. Whenever a section divided such a tract transversely a vessel could always be detected in its centre (Pl. XII, fig. 5). Where the infiltration was extensive the corpuscles were so densely massed that the individual cells could scarcely be distinguished (Pl. XII, fig. 7), but where it was slighter a vessel could often be seen to be encrusted with a layer of such cells. In some

places a few only lay in the perivascular sheath, but in other places other similar corpuscles lay outside the perivascular sheath and in the adjacent tissue. In the white substance in the neighbourhood of these tracts similar cells lay among the nerve-fibres, and in some places were so numerous as to occupy a larger area than the nerve-fibres (Pl. XII, fig. 7). In some places, indeed, the nerve-elements appeared to have been destroyed.

These cells were deeply stained with carmine, and still more deeply by logwood. They resembled closely white blood-corpuscles in size and appearance. (The measurements given above correspond closely to those of white blood-corpuscles in hardened tissues.) They were identical in appearance with the white blood-corpuscles which were seen here and there among the blood-discs within the vessels. In some sections the walls of the vessels appeared to contain similar cells, as if in the process of diapedesis. From these appearances we are inclined to regard these cells as being actually white blood-corpuscles, and the morbid change as a *leucocytal infiltration*.

In some parts, in which the cells were scattered among the nerve-fibres, some were no longer round, but were oval and even angular (Pl. XII, fig. 5), and from the angles of some of these, minute processes extended, while in the neighbourhood were other angular cells, the branches of which extended between the nerve-fibres. These were larger and far more abundant than any existing in the normal cord, and from the transitional forms they appeared to be in the process of development from the infiltrating corpuscles.

In many places near this infiltration there appeared to be an increase in the nuclei of the neuroglia, the interstices between the fibres being studded with minute nuclei, smaller than red blood-discs, and much more numerous than in health. In some parts this was noticeable apart from any leucocytal infiltration.

Where the infiltration had taken place into the grey

substance, the matrix of the grey matter had undergone a granular degeneration, or else presented an oedematous appearance, its constituent elements being separated by clear spaces, which gave it a spongy appearance.

The nerve-cells in many parts of the cord appeared perfectly healthy. In most places, however, they presented the same granular appearance which was noticed in the fresh state. Where the grey matter was invaded by the leucocytes, the nerve-cells were completely surrounded by them. Some of the cells had a remarkable appearance, their outer boundary was not well defined, the processes had disappeared, the substance of the cell was granular, the nucleus distinct, the cell-substance in the immediate vicinity of the nucleus stained deeply with carmine, that near the outer part of the cell much less deeply, and the indistinctness of the outer boundary gave the cell the appearance as if it were being dissolved (Pl. XIII, figs. 10 and 11). Cells presenting such an appearance were seen very near to cells which had fairly healthy characters, and the change cannot, therefore, be ascribed to any effect of the hardening agent.

In many parts of the cord the substance of the large nerve-cells was encroached upon by rounded vacuoles of various size (Pl. XIII, figs. 8 and 9). Sometimes these lay entirely within the body of the cell, but more frequently they were partly outside in some coagulable fluid in which they had apparently formed in the process of hardening. They were precisely similar to the vacuoles seen in the substance of albuminous exudations in other organs, as into the tubules of the kidney in acute Bright's disease. Many cells were almost destroyed by them. They are probably of pathological significance, even though of artificial origin, and may indicate the presence, in the vicinity of the cells, of the results of excessive action.

Anatomical description of lesions in the spinal cord.—Midway between the *first and second pair of cervical nerves* an extensive infiltration of cells has occurred into the

right posterior cornu in the position of the posterior vesicular column; the matrix of the nerve-tissue here is granular and broken up; the cells are scattered irregularly through it, and some are aggregated around a vessel. On the left side of the cord, vessels here and there were varicose. A few sections lower down similar infiltration and disintegration exist in the *left* cervix cornu posterioris, and in addition to the lymphoid cells, blood discs are infiltrated into the tissue.

At the level of the *second cervical nerves* lymphoid cells are scattered through the left anterior cornu, near the grey commissure, and are aggregated especially around a vessel in this position. In both right and left lateral columns is a scattered infiltration of cells, those on the left side occupying areas of considerable extent. There is no general thickening of the neuroglia.

Third cervical nerves.—Extensive cellular infiltration into the posterior column and into both lateral columns. The anterior columns are perfectly free. In the right lateral column the infiltration is confined to the anterior half, the posterior half being quite free. In the posterior columns the infiltration is in places very dense, and the individual cells cannot always be recognised in the thickened tracts which extend through the white substance. In some places these appear as if consisting of fibrous tissue, but on the margins of these tracts the separate round cells are visible. In many places the perivascular sheaths of the vessels are seen to be distended with these cells. In other places vessels running transversely appear covered with a cellular incrustation. In the lateral columns, besides this cellular infiltration, there is a considerable increase in the minute nuclei between the nerve-fibres. The left caput cornu posterioris is also infiltrated with lymphoid cells, and a similar infiltration extends along the posterior nerve-roots to the surface of the cord. The pia mater is here and there separated from the surface of the cord by a structureless exudation.

Fifth cervical nerves.—The left anterior cornu appears

to be literally crammed with leucocytes throughout its anterior half, and from it tracts of similar cells extend along the connective-tissue trabeculæ into the adjacent anterior column. Small areas of granular débris exist here and there in the anterior cornu. In the anterior column adjacent to the cellular infiltration there is much increase in the nuclei of the neuroglia, and this increase, in a slighter degree, is to be noticed throughout all the white columns.

Sixth cervical nerves.—The right anterior cornu is here full of leucocytes, and is distinctly enlarged by their infiltration. Its width is just one third more than the left anterior cornu. The infiltration is especially dense in the superficial layers of the grey matter near the white column, and also in the adjacent white substance, and the vessels which pass from the surface through the anterior column are surrounded by cells. The central part of the anterior cornu appears as if in a state of œdema. Instead of a finely granular matrix it has a spongy appearance; there is a network of fine fibrillæ, but these are separated by clear spaces. In the anterior part the large nerve-cells appear swollen and somewhat granular, and are surrounded by leucocytes. The same appearance of œdema may be seen in the adjacent white column; clear spaces exist between the trabeculæ of connective tissue, spaces in which no nerve-fibres or axis-cylinders can be seen.

First dorsal nerves.—The central canal of the cord was widely distended by granular exudation. The posterior columns were densely infiltrated with lymphoid cells, especially the outer half of the right posterior column. In the superficial layers of the posterior columns there is a considerable increase in the neuroglial tissue, almost all the axis cylinders having disappeared, and a nucleated connective tissue remaining in this place.

Second dorsal nerves.—The right half of the cord is fairly healthy, but the left presents extensive changes. The anterior column and the adjacent part of the lateral column present a dense cellular infiltration, and there is also

some infiltration of the grey substance of the anterior cornu.

Third dorsal nerves.—The cord is fairly healthy. A small number of lymphoid cells lie along the vessels in the lateral column near the right posterior cornu, and the vessels of the grey substance of the posterior cornu have the appearance of the walls being crammed with similar cells.

Fourth dorsal nerves.—The left anterior column, between the anterior cornu and the surface, presents numerous tracts of cellular infiltration. Towards the surface these are very dense, and occupy the whole tissue. The nerve-fibres in the anterior column are quite distinct, but towards the lateral column they become separated by the infiltration of lymphoid cells, and finally the nerve-fibres disappear, and are replaced by irregular masses of myelin, partly fused together and lying between the dense tracts of cells; here and there an axis cylinder can still be seen. In several other spots in the neighbourhood of the infiltration the nerve-fibres have disappeared and have left large spaces. The adjacent part of the anterior cornu is crammed with cells in its entire width, and one or two nerve-cells are granular and swollen so as to be globular in form, with very indistinct processes. They appear as if in process of disintegration, as already described.

Fifth dorsal nerves.—The cord presents scarcely any deviation from the natural state, the only change being the presence of tracts of lymphoid cells along the vessels in the posterior column.

Eighth dorsal nerves.—The cellular infiltration is chiefly in the posterior column and in the anterior part of the left lateral column. The anterior cornu presents the œdematous appearance already described. The nerve-cells have here undergone extensive vacuolation, clear spherical spaces existing in and outside them as already described.

Tenth dorsal.—The infiltration is chiefly into the middle of the left half of the grey substance, near the

cervix cornu posterioris; the posterior cornu is also densely infiltrated with cells. These extend along the posterior nerve root, and at the extremity of it are accumulated at the surface of the cord. There is also much infiltration into both posterior columns, and into the anterior part of the right lateral column.

A few sections lower down the infiltration is chiefly into the posterior parts of the two lateral columns, outside the posterior cornua, and it extends to the surface of the cord where are large aggregations of myelin.

Eleventh dorsal nerves.—The infiltration chiefly affects the right half of the cord, involving mainly the peripheral portion of the anterior columns and the posterior part of the lateral columns. A slighter degree of infiltration extends throughout the whole anterior column as far as the grey substance.

About the twelfth dorsal, and between it and the first lumbar nerves, there are enormous aggregations of leucocytes in the right half of the posterior column, and also in both antero-lateral columns. A vessel which passes from the surface into the anterior column is covered with a dense coating of cells exactly similar to those which can be seen here and there within the vessel. Similar cells are infiltrated into the adjacent tissue, but gradually decrease in number away from the vessel. There is also much infiltration of the inner half of the right anterior cornu; the cells being in places aggregated around vessels, but for the most part scattered uniformly through the grey substance. The large nerve-cells appear very granular. There is also much irregular thickening of the pia mater over the posterior columns.

Second lumbar.—The chief infiltration is in the left anterior column, but there are some dense masses of cells in both anterior cornua, especially in the outer portions. Around one of the vessels which are seen in section the cells form an aggregation about four times the diameter of the vessel. The walls of the vessels appear somewhat thicker than normal.

Third lumbar nerves.—The central canal is considerably distended, and in its centre is an aggregation of lymphoid cells. The large multipolar nerve-cells appear swollen and granular, with indistinct outlines as already described. The vacuolation is very conspicuous, and in many cells has proceeded to the almost entire destruction of the cell with the exception of the nucleus. Near the cells which appear in a state of disintegration, vessels are seen which are surrounded by lymphoid corpuscles. In the white column, near to the grey matter, are two large oval masses tinting deeply with carmine; in their outer portions round cells are seen resembling in every respect those infiltrated elsewhere; in the centre of the mass, however, only a reticulate structure can be seen, consisting of branching cells which have oval spaces between them. The appearance suggests the idea that these branching cells have developed from such lymphoid cells as are seen on the margin. In the left lateral column a similar change has occurred. There is a considerable increase in the interstitial fibrous tissue, which, in some spots forms, imperfectly fibrous tracts of some size, and in this tissue are many large nuclei resembling the nuclei of the infiltrating cells.

Fourth lumbar nerves.—The round cells are uniformly infiltrated through the left anterior grey cornu, and are in places aggregated around the nerve-cells. The right anterior cornu is in its outer half, and the adjacent part of the lateral column quite destroyed by the invasion of these cells. There is also much destruction in the outer part of the lateral column, where only a few nerve-fibres can be seen. The rest are blended together into a uniform granular degenerated mass, not tinting with carmine or with logwood. In the inner part of the column the outlines of all the nerve-fibres have disappeared, but the axis-cylinders can still be traced among the cells with which the area is occupied, and which are aggregated very densely along some vessels crossing this part, and just outside the grey substance a vessel so

covered with leucocytes runs across a clear empty space, which has apparently resulted from the disintegration and destruction of the nervous and connective tissue. The inner half of the posterior cornu and the adjacent external tract of the posterior column is the seat of a dense infiltration, uniform in the cornu, but in the white substance distributed in tracts along the vessels. The adjacent fibres of the posterior median column seem to have perished and are replaced by masses of myelin, in which no axis-cylinders can be seen. At the spot thus degenerated there is but little cellular infiltration. A few sections lower down, the infiltration in the posterior cornu becomes much more dense.

A little lower, between the fourth and fifth lumbar nerves, the grey matter presents the same infiltration; there is much less disease in the posterior column, but the interstitial connective tissue is much more abundant than in health, and contains large numbers of minute granule-like nuclei. Almost every nerve-cell in the anterior cornu presents the same granular appearance and indistinct outline already described. Their nuclei and nucleoli remain distinct. A few large nerve-cells near the posterior vesicular column appear much swollen, and their nuclei cannot be seen. The other columns of the cord are fairly healthy.

A few sections lower down there is a wedge-shaped area, having its base to the surface, in which all nerve-elements have disappeared, and all that remains is a reticulate connective tissue, consisting of elongated fibres or fibre-cells. In the middle of this tract a vessel runs.

Fifth lumbar nerves.—Infiltration exists in the posterior column and posterior part of the lateral column.

A little lower there is less infiltration, but some exists around the vessels in the anterior columns, and there are a few tracts of cells in the posterior columns. Some nerve-cells appear to be apparently healthy, others appear to be swollen and disintegrating.

A few sections lower down the infiltration of the

posterior column is very extensive, and tracts extend on each side of, and parallel to, the posterior median fissure, from the surface of the cord almost to the posterior grey commissure, and from them branching tracts of infiltration extend on each side.

In the *conus medullaris* there is less disease; the most conspicuous change is an infiltration around the vessels in the anterior columns.

Medulla oblongata.—Changes very similar to those found in the spinal cord could be found scattered irregularly through the medulla, the chief change being the aggregation of lymphoid cells in the neighbourhood of vessels, such as has been just described. This change in the medulla was found chiefly in the white columns, and was much less extensive than in the spinal cord.

In the lowest part of the medulla, above the first pair of cervical nerves, the right anterior column was densely infiltrated with cells, some of which surrounded, and here and there concealed, some of the bundles of fibres of the nerve-roots. In the right lateral column were some irregular broad areas, adjacent to the vessels, packed with cells. A few sections higher, the position of a vessel in this column was marked by a large mass of cells, the aggregation being $\frac{1}{30}$ th of an inch long and $\frac{1}{150}$ th broad. It appeared to consist entirely of round lymphoid cells mixed with much granular matter.

The anterior pyramids at and just above the decussation were healthy, but just outside the left pyramid was a slight cellular infiltration. Just above the decussation the left posterior pyramid was the seat of extensive infiltration throughout its entire extent. The right was normal. There was slight infiltration of the right lateral column, and a mass of cells lay in the course of some of the fibres of the left hypoglossal nerve. At the lower portion of the hypoglossal nucleus there was extreme disintegration of the grey matter around the central canal; the substance was riddled with minute vacuoles separating the nerve-elements. Higher up the hypoglossal nucleus was normal.

In the upper part of the medulla the changes were very slight, being confined to a slight infiltration of the upper portion of the restiform bodies, and a few dilated vessels, occasionally with slight neighbouring cellular infiltration, near the floor of the fourth ventricle.

Cerebellum.—The changes found in the cerebellum were scattered irregularly through all the principal divisions of the organ, so that it was difficult to say that one part was more diseased than another.

The medullary centre presented, scattered through it, alterations similar in character to those which existed in the white substance of the cord. These consisted in the infiltration of the tissue with minute round cells (Plate XIII, fig. 14), for the most part aggregated around the vessels, but in some places scattered through the intervening tissue. In almost every process of medullary substance more or less infiltration could be traced, but the densest change was in the white centre itself, in which here and there tracts of tissue were entirely occupied by this cellular infiltration. The cells were for the most part round, and closely resembled those of the infiltration into the cord, and also those of the adjacent granular layer of the cerebellar cortex. In some, distinct nuclei could be seen. Some cells, as in the spinal cord, were angular and fusiform. Where this infiltration was abundant, the nerve-fibres of the medullary centre were separated by clear spaces, an appearance precisely similar to that seen in some parts of the spinal cord, and which has been spoken of as suggesting a condition of "œdema."

The layers of the cortex were for the most part healthy; here and there, however, slight changes were apparent. The granule layer was little affected; the only alteration was that in some places the nuclei were separated by clear spaces, or by small collections of yellowish débris, which resisted the carmine. The large corpuscles of Purkinje were for the most part healthy, but in places presented changes similar to those in the large nerve-cells of the cord. They were granular, slightly swollen, surrounded

by slightly granular débris, and vacuoles existed partly in adjacent, finely-granular material, and partly in the substance of the cells (see Plate XIII, fig. 13). The intervening substance of this layer had in places a degenerated appearance, the tissue elements being separated by minute clear spaces. The external "grey layer," was also for the most part healthy, but here and there was a dilated vessel filled with blood. These were seen especially in the neighbourhood of the change just described in and around the cells of Purkinje. In one place an extravasation of blood had taken place into the most superficial portion of this layer.

CASE 2.—For an opportunity of examining the spinal cord in the second case we are indebted to Dr. Hughlings Jackson, who has published a note of the symptoms presented by the dog, in the 'Lancet' for May 1st, 1875.

The movements in this case were confined to one fore-leg. Unfortunately we have been unable to learn which fore-leg was affected. The animal was killed by pithing, under chloroform, and after the spinal cord had been divided the animal lived for a few minutes, and during those few minutes the movements in the fore-leg were observed to continue.

An examination in the fresh state revealed no embolism of small arteries, and no obvious disease of the encephalon or spinal cord. The cord and medulla alone were examined with the microscope.

The *medulla oblongata* was healthy throughout, with the exception of slight damage to the lowest part of the right anterior pyramid, caused apparently by the pithing.

Spinal cord.—The highest portion of the cervical cord was quite healthy. Elsewhere throughout this region the nerve-cells of the anterior cornua were very granular, and appeared somewhat swollen, in some instances so that the body of the cell had an almost globular form. This change in the cells was much more marked in the

right anterior cornu than in the left, and was almost confined to the middle and lower parts of the cervical enlargement. The granules in some cells were so large and refracting as to appear like spherules of fat (Plate XIII, fig. 12). At the highest part of the cervical enlargement the edge of the anterior column adjacent to the anterior median fissure was for a short space irregularly infiltrated with minute nuclei, each about $\frac{1}{7000}$ inch in diameter, exactly resembling those normally existing in the neuroglia. In places in the right anterior cornu round cells could be seen adjacent to the vessels, apparently an early stage of the cellular infiltration so abundant in the spinal cord just described.

The upper dorsal region of the cord presented nothing abnormal. Below the middle of the dorsal region there was some symmetrical disintegration of the posterior cornua, especially of their outer portions, and at one spot the disintegration extended across the posterior commissure.

In the upper part of the lumbar region the nerve-cells of the right posterior vesicular column were swollen and granular. The difference between the cells of the right and left columns was very marked.

In the mid-lumbar region the left anterior cornu contained some much dilated vessels. A greatly dilated vessel ran along a nerve root through the right anterior column, and outside the vessel was a small extravasation of blood.

The lowest part of the lumbar region presented no abnormality.

REMARKS.

The striking difference in the minute changes found in the two cases renders it difficult to draw any inference as to the morbid condition on which the disease depends. It would appear from the symptoms that the disease in the second case (in which one fore-leg only was effected) was in an earlier and slighter stage

than in the first case, since in the latter one fore-leg was affected in the first instance, and the spasm afterwards spread to other parts. In the slighter case the only morbid appearance at all conspicuous was the granular and swollen condition of the nerve-cells. The other changes noted were slight and limited in area. This condition of the nerve-cells is one which might well have resulted from such overaction as the clonic spasm necessarily involves. There was evidence of vascular turgescence only here and there. In the other cord a morbid state of the nerve-cells was also apparent, somewhat different from that in the slighter case, but very conspicuous, and also of such a character as may reasonably be ascribed to their overaction. But in this case other extensive lesions were found, consisting of a cellular infiltration related in position to the vessels, and which it seems reasonable to ascribe to a vascular disturbance. This lesion occupied not only the cord but the medulla oblongata and cerebellum, while the corpora striata and hemispheres were free from it.

Two views may be held as to the relation of this cellular infiltration to the disease. It may be regarded as primary, and the cause of the irritation of the nerve-elements, or, as secondary to this overaction, to the excessive functional activity of the cerebellum and cord. There are difficulties in the acceptance of either explanation, and the question can probably be decided only by further observations. On the one hand, the distribution of this change was apparently random, conspicuously unsymmetrical, and in no way related to the function of the parts. (In this respect it presents a marked difference from the vascular changes found by Dr. Dickinson in the chorea of man.) From its distribution and extension it seems clear that this cellular infiltration, to whatever cause it may have been due, ran an independent course and produced its own effects. In different places the posterior columns (outer and inner fasciculi), the grey matter (anterior and posterior horns and central part), the

anterior and lateral white columns, were all affected. The infiltration must have interfered with the function of the structures in which it occurred; in some places, where it was most intense, destruction of the nerve-elements had taken place. The loss of power and the loss of sensation may, with probability, be ascribed to the effect of this local infiltration. On the other hand, the wider limitation of this change to the cerebellum, medulla, and cord, makes it difficult to believe that it can have been due to a primary lesion of the vascular system (such as embolism or thrombosis). Another difficulty in accepting such a view is that we should have to assume a different pathology for the two cases, since in the slighter case no such cellular infiltration or evidence of considerable vascular disturbance was found. It would seem, therefore, most in harmony with the facts of the two cases to assume that in its origin this cellular infiltration depended on a vascular disturbance, which was not primary and independent, but was secondary to the overaction of the nerve-elements. Once set up, the cellular infiltration continued, increased, and produced its own consequences.

It is to be noted that in some places the minute infiltrating cells were becoming angular and fusiform in those parts of the infiltration which appeared of oldest date. This change suggests the probability that the cells may become transformed into connective tissue, and that a condition of disseminated sclerosis may result from this scattered infiltration.

The effect of section of the cord, in the two cases, was singularly at variance. In the one case, in which very slight lesions were found in the cord, the movements persisted after section. This is in accordance with the observations of Chauveau, Onimus, and others, who have found that in similar cases the movements have usually persisted after the section. But in the case in which extensive spinal lesions existed, section of the cord arrested the movements, except in those muscles which are supplied by the medulla oblongata. From this fact

the inference might be drawn that the movements depended on the encephalic changes, and that the section arrested the transmission of the morbid action. But this conclusion is contradicted by the persistence of the movements after section of the cord in other cases in which the movements resembled closely those in the case in which they ceased. We should have to assume that movements of the same character were, in the one case spinal, in the other encephalic in their origin, and that they were encephalic in the case in which most extensive spinal lesions were found. A more probable explanation of the cessation of the movements is that they were inhibited by the shock of the division of the cord. The nerve-cells may have been weakened by their long-continued over-action, so that their inhibition was more easily produced than in the slighter case in which the disease was in an earlier stage. This is rendered highly probable by an observation of Onimus and Legros,¹ that in one case the movements ceased for five minutes after section of the cord, and then recommenced.

Other observations will be needed to settle these and some other points of interest which the cases suggest. They show, at any rate, that it is important that the nerve-centres should be carefully examined in conditions of disordered nervous action in the lower animals.

¹ 'Electricité Médicale,' 1872, p. 383.

DESCRIPTION OF PLATES XII AND XIII.

The Pathological Anatomy of Canine "Chorea" (W. R. Gowers, M.D., and H. R. O. Sankey).

PLATE XII.

(Case 1.)

Fig. 1. Spinal cord, lumbar enlargement; dense infiltration into right half of anterior grey cornu and adjacent part of lateral column.

Fig. 2. Ditto, lower dorsal; infiltration into left lateral column, and right anterior cornu.

Fig. 3. Upper dorsal; infiltration into both lateral and posterior columns.

Fig. 4. Posterior columns; branching tracts of infiltration.

Fig. 5. Section of vessel with infiltrating cells, round and angular, in vicinity.

Fig. 6. Healthy white column.

Fig. 7. White column, infiltrated with lymphoid cells.

PLATE XIII.

(Cases 1 and 2.)

Figs. 8—11, Morbid appearances of nerve-cells of anterior cornu, spinal cord (8 and 9, vacuolation; and 10 and 11, granular disintegration). Case 1.

Fig. 12. Granular nerve-cell, right anterior cornu of spinal cord, cervical region. Case 2.

Fig. 13. Cerebellum; (Case 1) granular state of cells of Purkinje, with vacuoles partly adjacent, partly in protoplasm of cell; distended vessel passing through grey layer and into granule layer.

Fig. 14. Medullary centre of cerebellum; (Case 1) infiltration of cells adjacent to vessel.

FIG 1.



FIG 2.



FIG 3.

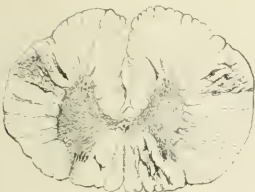


FIG 4.



FIG 5.

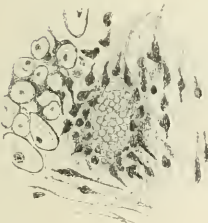


FIG 6.

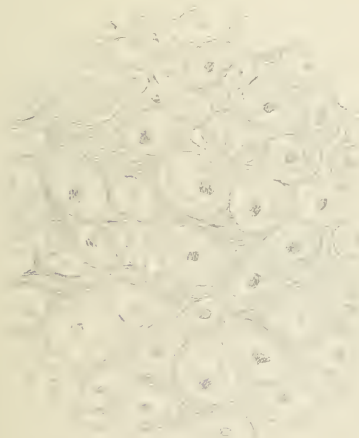


FIG 7.

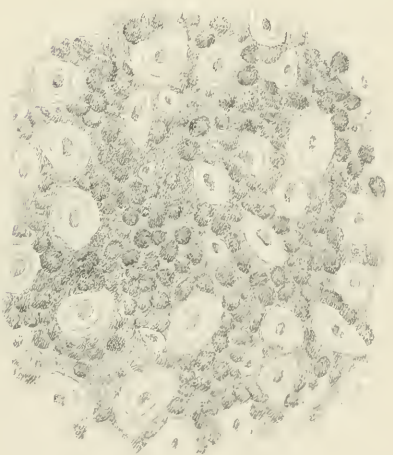


FIG. 8

FIG. 9

FIG. 10

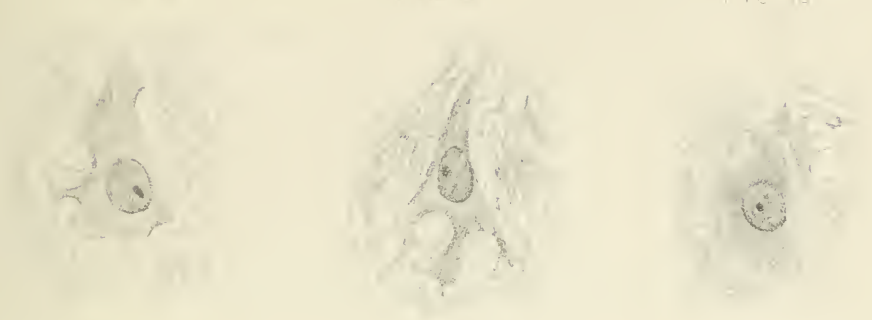


FIG. 13

FIG. 12

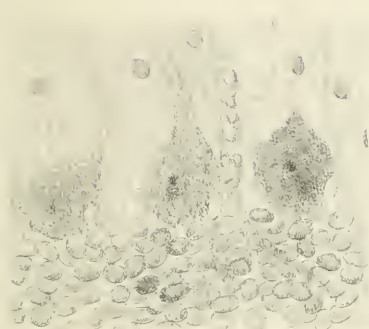
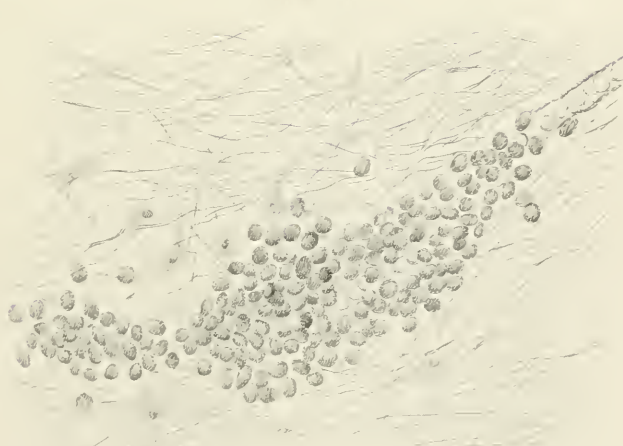


FIG. 14

FIG. 11



ON

DIRECT WOUNDS OF THE URETER.

BY

TIMOTHY HOLMES, F.R.C.S.,
SURGEON TO ST. GEORGE'S HOSPITAL.

Received March 2nd—Read April 10th, 1877.

THE following case is, I think, worthy of the attention of the Society, in connection with a subject which has been little studied in this country, viz. direct wounds of the kidney and ureter. The case, indeed, is not free from ambiguity, which will be fully pointed out in the sequel. The prompt recovery of the patient, fortunately, precluded all possibility of confirming the diagnosis by anatomical examination, and the character of the discharge from the wound was very different from that of healthy urine. Yet I hope to prove to the satisfaction of the surgeons who may think the following case worthy of their attention, that the source of the copious watery discharge which followed the stab in this instance must have been the urinary tract; and that it proves the possibility of a direct and uncomplicated wound of the ureter through the posterior parietes of the abdomen.

CASE.—The patient, Hugh M—, æt. 13, was stabbed by accident by another boy on January 7th, 1877. The

author of the mischief was running up a staircase after his play-fellow, holding an open clasp-knife in his hand, and half-unconsciously drove the blade into his back. The knife (as was proved by a subsequent examination of the patient's clothes) passed through his jacket, which was a long one, on the left side of the seam, the end of the cut just touching the seam; but it passed through the trowsers on the right of the seam, glancing *upwards* and *outwards*, *i. e.* across and then *away from* the middle line. It entered the boy's body just on the right of the middle line, and about on the level of the posterior superior spine of the ilium. The wound bled a good deal, and Dr. Forsyth, of Greenwich, was at once sent for, and arrived about half an hour after the injury. The bleeding had then stopped, and Dr. Forsyth was about to apply a compress to the little wound, which was not much above a quarter of an inch long, when he was surprised to find that drops of clear fluid were oozing out of it. Touched with nitrate of silver this fluid gave a white precipitate. The boy was put to bed, and the fluid ran freely from the wound during the whole night. Next day he was suffering from nervous symptoms, viz. faintness, pain in the head, and stiffness of the fingers of both hands. He had occasional vomiting. The respiration was sighing; the pulse 130. He passed by the urethra during the day, about 20 oz. of urine, which was high coloured and contained a large quantity of pink urates, but no blood or albumen.

I saw him the following day (January 9th), in consultation with Dr. Forsyth. He was very much better, had vomited once after food, still complained of slight headache, and the fingers were still a little cramped, but not much. The pulse was 86, and he was quite free from fever, the temperature being only a little above the normal. From the little puncture in the back perfectly clear fluid was still oozing continuously, soaking the bed-clothes through the draw-sheet. There was a decided smell of urine in the bed. Dr. Forsyth reported that he

had passed a probe into the wound for about an inch on the previous day, and that it led in a direction upwards and outwards. This being so, I did not think it desirable to repeat the examination. A sponge, which had been laid beneath the wound, was squeezed out and some of the fluid thus obtained from the wound, as well as some of the urine passed by the urethra, was taken away for examination. As the symptoms were so slight in themselves, and the first effects of the injury were evidently passing over, we felt justified in giving a good prognosis, though we thought it possible that if the fluid were urinous, as we believed it to be, a fistula might remain. We ordered a draught containing hyoscyamus and bromide of potassium, and advised the friends to persuade the boy to lie on his face as much as possible, but he was too restless to maintain this position for any length of time. We did not think it advisable to make any attempt to close the wound.

It is not necessary to give the daily notes taken by Dr. Forsyth for the next few days. The fluid continued to run out of the wound in immense quantities. It was perfectly impossible to measure the quantity, but it was ascertained that for several days together three large draw-sheets, which were placed under the wound, and which when wet through in one part were shifted to another, were thoroughly soaked in the course of the day with the fluid which ran out of the puncture. Meanwhile his headache and nervous symptoms diminished steadily, and he went on well in all respects.

On the 13th it is noted that the wound had closed externally for a time, and that the bedding was far less wet. On inspection, the closed wound was seen as the apex of a cone, formed by the fluid raising the surrounding tissues. On introducing a probe, about $\frac{3}{4}$ of the confined fluid gushed out to a considerable distance. After this the discharge of the fluid continued as copious as ever till the 20th. He was very restless on the night of the 19th and in a copious perspiration, complaining of

backache and headache. About 10 a.m. the wound became entirely obstructed, but, on opening it with the pointed end of a probe, a stream was projected to the distance of three inches. Nearly 3 oz. of fluid thus escaped.

The wound finally closed during this night. It was seen to be open and discharging after 11 p.m., but was closed at 6 a.m. He suffered a good deal from headache, and somewhat from vomiting, during this period.

From this time there was nothing to note. I saw him last on January 30th, and heard of him some weeks afterwards perfectly well.

I append notes of the examination of the urine passed by the urethra and of the fluid which ran from the wound, by Dr. Forsyth, and Dr. Ewart, Curator of the Museum of St. George's Hospital, and a single examination of the fluid from the wound by Dr. Ralfe, Lecturer on Physiological Chemistry at St. George's Hospital. I should add that after the first specimen, which was squeezed out of a sponge, the fluid from the wound was obtained quite pure.

First, with regard to the urine passed naturally, Dr. Forsyth noted its quantity from day to day, and found it to vary from 16 oz. in the twenty-four hours, on January 11th (the first exact note as to quantity after the accident), to 25 and even 40 oz. some days afterwards. On the closing of the wound, the quantity was found to be between 46 and 36 oz. The exact statements from day to day of quantity and specific gravity are appended.

Quantities of urine passed by the urethra, with dates and sp. gr.

| | | Sp. gr. | |
|-----------|-------|---------|--|
| Jan. 11.— | 3xvj | ... | 1030. |
| „ 12.— | 3xxv | ... | 1026. |
| „ 13.— | 3xl | ... | 1020-30. Wound closed. Opened. |
| „ 14.— | 3xx | ... | 1027-30. |
| „ 15.— | 3xxiv | ... | 1030. |
| „ 16.— | 3xxx | ... | 1020. "Inclined to close" (Miss M—'s expression at the time), but sheets wetted. Opened. |

| | Sp. gr. |
|----------------------------------|---|
| Jau. 17.— $\tilde{z}xvij$... | 1024-33. |
| „ 18.— $\tilde{z}xxj\dagger$... | 1030. |
| „ 19.— $\tilde{z}x\dagger$... | 1029. |
| „ 20.— $\tilde{z}xix\dagger$... | 1030. Wound closed for 4 hours. Opened. |
| „ 21.— $\tilde{z}xlvj$... | 1029. Finally closed. |
| „ 22.— $\tilde{z}xxxvij$... | 1015-25. |
| „ 23.— $\tilde{z}xxv$... | 1020-27. |
| „ 24.— $\tilde{z}xxxij$... | 1018. |
| „ 25.— $\tilde{z}xxxvj$... | 1017-23. |

The sign \dagger implies that an unascertained quantity passed with motions, say from $\tilde{z}v$ to $\tilde{z}vij$.—A. F.

The urine was first noticed to deposit urates on January 9th, the day on which I first saw him, and this deposit continued ever afterwards while the wound was open, and even for some time after its closure, for when I last saw him, on January 30th, there was still a copious deposit of pale urates. No pus or blood was ever noticed in the water, except on the 17th, four days before the final closure of the wound, when a very small quantity of blood was visible to the naked eye, and, as will be seen below, a microscopic trace of blood remained for some time afterwards. The specific gravity (as seen by the appended notes) varied considerably. The first quantity tested after the closure of the wound (January 22nd) had a sp. gr. of only 1015, which is worth noting, and its reaction was very faintly acid. The usual reaction of the urine was decidedly acid, but this varied. Occasionally, though very rarely, it was alkaline.

The following are Dr. Ewart's notes of the examination of fluid from the wound and specimens of urine.

A. Analysis of fluid squeezed from the sponge which lay on the wound January 9th :

Sp. gr. 1017. Good crystals of nitrate and of oxalate of urea were obtained from it, and a feeble uric acid reaction. There was not enough remaining to test for sugar.

B. Analysis of fluid from the wound, collected quite free from any possibility of foreign admixture :

Colourless, odourless. Reaction neutral. Sp. gr. 1008. No albumen. Under the microscope no casts, blood, epithelium, or crystals—only bacteria and dirt.

Evaporated to dryness in a water-bath, the fluid left a small residue, non-crystalline, nearly hyaline, devoid of smell. This was taken up with a little distilled water :—
i. A few drops allowed slowly to evaporate on a microscopic slide gave an abundant crop of chloride of sodium crystals, but no crystals of urea. ii. Another portion was mixed with nitric acid. No nitrate of urea crystals could be detected. iii. The same process with oxalic acid gave a negative result. iv. A fair portion of the residue left after careful evaporation was subjected to the murexide test. No coloration was obtained either with potash or ammonia. v. Sugar was tested for with Fehling's solution. None was found.

The urine of the same day was tested, and found to be of sp. gr. 1030, faintly acid. Colour pale yellow, containing [no albumen, casts, or blood, but an abundance of uric acid crystals and small crystals of oxalate of lime.

Another examination of the fluid from the wound on January 18th gave results identical with that of January 14th.

c. Another comparison of the fluid passed from the wound and of the urine of the same day was made on January 20th :

The fluid from the wound was colourless, odourless, and slightly turbid. Reaction feebly alkaline. Sp. gr. 1008. No albumen. No crystals. Complete absence of organised sediment, except bacteria. The methods described above were again applied, with negative results. No urea, no uric acid, and no sugar were found. Then the experiments were repeated with the following additional precautions. The original residue was treated with absolute alcohol. The residue formed from the alcoholic extract was redissolved in distilled water, and this solution allowed to evaporate slowly in three portions—(1) alone, (2) with nitric acid, (3) with oxalic acid. Nowhere was

any trace of urea evident. The residue, examined chemically, revealed the presence of abundant chlorides, a small amount of sulphates, and a mere trace of phosphates.

The urine of the same day was of a dark-yellow colour. Sp. gr. 1032, containing a small sediment of blood, and showing a haze of albumen. It contained uric and oxalate of lime, but no casts.

Dr. Ralfe reported with regard to another specimen of the fluid from the wound,¹ that it was alkaline, containing $\cdot 08$ parts of solid to $9\cdot 92$ of water; that the solids consisted of an ordinary albumen, coagulable by heat, and an albumen not so coagulable, but precipitable by acetic acid; that as to fats there was a slight greasy residue from evaporation of the ethereal extract of the dried residue, but no cholesterine; that as to extractives there were traces of urea, $\cdot 007$ in 10 parts, but no uric acid and no glucose; and as to salts there was chloride of sodium $\cdot 005$, and phosphates $\cdot 003$, in 10 parts.

I have been particular in giving the details of this case, inasmuch as I cannot, by search in surgical works or inquiry among my colleagues, find one at all similar to it.

The chief question connected with it is the source of the discharge. There are only, as far as I can see, two possible alternatives. The fluid must either have been urine from a puncture of the ureter, or cerebro-spinal fluid from a puncture of the spinal membranes, through the ligamenta subflava. It seems impossible that the kidney itself could have been wounded; first, because the wound was much too low, considering the length of the knife-blade; and, secondly, because of the absence of any blood in the urine immediately after the injury, or any serious hæmorrhage from the wound. A wound or laceration of the renal substance always, as is well known, gives rise to copious hæmorrhage. Gustav Simon, who has written an interesting treatise on the surgery of the

¹ I have not noted when this fluid was obtained, but I believe it to have been subsequent to that last examined by Dr. Ewart.

kidney,¹ in which he describes ten cases of wound of that organ, says that hæmaturia is an invariable symptom. And we all know what copious hæmaturia follows on bruises or laceration of the kidney. In this case there was no hæmaturia, except to the most trifling extent, and not till long after the accident. And the appearance at that time of a few drops of blood in the urine is, as I shall point out by-and-bye, confirmatory to some extent of the view which I take of the case, that the ureter was wounded.

I may also dismiss any such wild supposition as that there was some cyst in the lumbar region which had been punctured. There was no trace whatever of any tumour. Nor could a cyst, after having been punctured, have continued to furnish so enormous a flow of perfectly clear fluid for a fortnight.

There remain, then, the two alternatives above mentioned, and though the diagnosis may not be absolutely certain, I submit that it is far more probable that the ureter was the seat of injury.

I place before the Society a preparation to show how easily a knife, made to enter (as in the case of my patient) close to the posterior superior spine of the ilium, can be driven inwards into the spinal canal through the ligamenta subflava, or outwards and upwards through the psoas muscle into the ureter. This preparation was made from the body of a young child. In the body of another child, about the same age as my patient, I drove a narrow knife, as nearly as I could guess, in the direction in which the wound was thought to have run in the case before us. I felt one of the transverse processes as the knife passed in. Having passed a probe along the knife I withdrew the latter, and then, on dissection, found the point of my probe lying just underneath the peritoneum, within a quarter of an inch of the ureter. The kidney had been previously

¹ 'Die Chirurgie der Nieren.' Von Dr. Gustav Simon, Prof. d. Chir. in Heidelberg. Stuttgart, 1876.

removed, but I noted that there was at least an inch of the ureter left above the point of the probe.

We may at once admit, then, the possibility of a wound of the spinal membranes from such an accident, and they are of course nearer the skin than the ureter is, though the latter is, I am sure, fairly within the reach of the knife which caused this accident. But the difficulties in the way of regarding this fluid as cerebro-spinal are, I think, insuperable. In the first place the examination of the child's clothes, and Dr. Forsyth's examination of the wound, render it nearly certain that the knife was directed *outwards* and upwards, while in order to reach the spinal membranes it must have passed inwards. Then, how can we conceive that the membranes could be laid open, yet none of the cords of the cauda equina be injured (which in this case they assuredly were not), nor any symptoms of spinal inflammation follow, during the whole fortnight that the wound remained open? Again, could the cerebro-spinal fluid be secreted in sufficient quantity to furnish this enormous flow. I speak on this point with some diffidence. I once assisted Mr. Athol Johnstone to remove a congenital tumour formed in the interior of the spinal canal in the sacral region, the laminæ being unossified. On the removal of the tumour the membranes were fully exposed, and we saw them exhibit the same movements, synchronous with respiration, which the cranial dura mater is so often seen to present.¹ That case proves that there is a considerable quantity of cerebro-spinal fluid at the end of the canal. But here, the flow was so instantaneous, so equable, and so continuous, that it seemed as if it could only come from a perennial source of supply like the kidney. For surely the spinal membranes, if called upon at once for something so much beyond their usual quantity of secretion, even allowing that they could respond to the call (which is doubtful), must have exhibited some sign of the

¹ The case and dissection will be found in 'Path. Soc. Trans.,' vol. viii, pp. 16, 28.

irritation by which alone they could have been so stimulated. But in this case there were absolutely no signs of irritation or inflammation of any of the spinal structures. It is true that the child suffered from headache, and that occasionally for the first few days after the accident his fingers were a little stiff; but both those symptoms were too trivial in themselves to be symptomatic of any grave injury, especially they could not be connected with injury in the lumbo-sacral region, and similar nervous symptoms had been noticed in this boy by Dr. Forsyth in previous illnesses.

In regard to the quantity and composition of the cerebro-spinal fluid Magendie is quoted by Hirschfeld and L  veill  ,¹ as estimating it in a man of middle height at 62 grammes = 2 $\frac{1}{4}$ oz. about, though he adds that in spinal atrophy the quantity may be six times as great. In 'Quain's Anatomy'² the quantity is given conjecturally after Majendie as varying from 2 drachms to 2 ounces. So that we may probably take about 2 ounces as a fair average. The composition is thus given in Hirschfeld:

| | | | | |
|--|---|---|---|--------------------------------------|
| Water | . | . | . | 98.584 |
| Albumen | . | . | . | .088 |
| Osmazone | . | . | . | .474 |
| Chloride of sodium and potash | . | . | . | .801 |
| Animal matter and phoshate of lime | . | . | . | .036 (probably a misprint for .056). |
| Carbonate of sodium and phosphate of lime | . | . | . | .017 |

Other observers, I believe, regard glucose as a constant component of this fluid.

It is added in Hirschfeld, "Surgical cases [by which I presume fractures of the base of the skull are intended] show that this liquid is rapidly resecreted, but its loss in considerable quantities always causes death."

It seems, then, from the notes given at pages 253—255, that the composition of the fluid from this wound, though, no doubt, much nearer that of the cerebro-spinal

¹ 'Traité du Syst  me Nerveux,' 1866, p. 21.

² 'Quain's Anatomy,' eighth edition, vol. ii, p. 575.

fluid than of healthy urine, yet differs in specific gravity, in minute composition, and in the absence of glucose. I may add, on Dr. Ralfe's authority, that the precipitate caused by acetic acid in this fluid was probably mucin, and that this strengthens the view that the fluid was derived from the urinary passages. And, still more, the enormous and continuous flow of liquid from the wound, far from inducing death, as Hirschfeld says it would if cerebro-spinal, gave rise to no symptoms whatever. The only difficulty in regarding the wound as situated in the ureter was the character of the fluid. It is, I allow, somewhat surprising that so very small a puncture should have remained freely open, considering the mass of muscles and fasciæ through which the knife must have passed to reach the ureter; but nearly the same difficulty exists in the case of the spinal membranes, which are covered by all the mass of fasciæ and muscles lying in the vertebral gutter. The perpendicular course of the muscular fibres furnishes, I suppose, in either case, a reason why a small wound may remain open. If such a stab were to reach the ureter, that tube would be found full of fluid; its vermicular contraction would expel the fluid in drops, so long as no obstruction existed, and in a stream whenever the flow had been temporarily barred, just as happened in this case, and the flow would be permanent, even in the absence of any irritation, just as this was. None of these characters would be present in a wound of the spinal membranes. The composition of the fluid, however, which Dr. Ralfe's and Dr. Ewart's analyses show, does certainly render this explanation of the case difficult. It is true that Dr. Ewart's examination of the first specimen of urine did give some urinous reaction, but only to a microscopical extent, and only such as might easily have come from an accidental contamination (with urine) of the sponge from which that fluid was squeezed. And it was ascertained that the sponge in question had actually fallen into the chamber-pot a month previously. It had, however, been carefully scalded

afterwards, and had been in use as a washing-sponge during the whole of the intervening time; so that it was hardly likely that any urine could still remain in it. At the time when this fluid was collected (when I visited the boy) the urinary smell was quite perceptible in the bed, though hardly so strong as it would have been from so copious a flow of normal urine. After this time, however, no urinary constituents whatever were detected in the fluid, with the dubious exception of the very small quantity of urea in the specimen analysed by Dr. Ralfe, a quantity, however, which was in Dr. Ralfe's judgment inconsistent with the idea that the fluid was cerebro-spinal. The want of colour in the fluid is also a very remarkable circumstance if it is looked on as urine from the ureter.

The dubious composition of the fluid has been, however, noted by Mr. Stanley in the second of his two cases of ruptured ureter, related in the 'Med.-Chir. Trans.,' vol. xxvii. The patient was under Mr. Vincent's care. Mr. Stanley says (page 10), "The fluid obtained from the abdomen was found to be albuminous, and to contain a small quantity of urea, but from the indistinct recognition of the healthy urinary salts in it a high chemical authority pronounced it not to be urine." In this case, the source of the fluid from the pelvis of the kidney was proved by dissection. I may just observe that in the other case given by Mr. Stanley (which was under his own care) the sp. gr. of the fluid was the same as in the fluid tested by Dr. Ewart, viz. 1008 (see page 254). Here, however, the constituents of the urine were perfectly recognisable. Both in Stanley's and Vincent's cases the colour was yellow.

That the secretion from the kidney may in abnormal states approach in composition to that of the fluid in this case is well known. Thus, in Dr. Day's translation of 'Simon's Chemistry,' vol. ii, page 234, there are some analyses of watery urine in nephritis, quoted from Becquerel, in which the quantity of solids was almost as low as in this instance, and in the chapter on hydruria or

polyuria in the same volume (pp. 305, 306), there are some cases still more in point, though the details are not sufficiently exact. Thus, in a man under the care of a physician named Stosch, it is said that the urine (passed in large quantity, four to six quarts daily) "contained no sugar, and scarcely a trace of urea or of the other ordinary constituents of urine," words which are almost exactly applicable to our case.

A confirmation of the diagnosis is afforded by the condition of the urine passed by the urethra after the injury. We may assume that it was normal before the accident, as the child was in perfect health. As soon as it was examined after the injury it was found to be in very diminished quantity, and soon began to contain a very large quantity of lithates. After the first closure of the wound (on January 13th) the urine recovered its normal volume, fell again when the wound was reopened, rose again at the second closure of the wound, and recovered its natural quantity as soon as the wound had permanently healed.

At one time, shortly before the healing of the wound, a little blood appeared in it, a very natural result of any slight movement of the body while the granulations of the wound were in close proximity to the opening in the ureter, but a phenomenon quite inexplicable on the hypothesis of a wound of the spinal membranes.

If it could be shown that a wound of the ureter or a lesion of that organ could suspend the true secreting function of the corresponding kidney, while it left its percolating function intact, or even if any theoretical explanation of such a result could be given, the case would be quite clear, since the opposite kidney would have double secretive work to do, and the urine passed by the urethra would be scanty, with excess of lithates, &c. It is true that Stanley's cases go some way towards showing the possibility of this, but they can hardly be regarded as decisive, and in them, as the fluid was extravasated into the tissues and retained there for a

considerable length of time before it was drawn off, its composition (even if it had been more accurately ascertained than it was) might have been expected to differ from what it would have been if it had escaped directly out of a wound.

Lesion of the nerves of the kidney produces watery urine. "Section of the renal nerves," says Dr. Michael Foster,¹ "is followed by a most copious secretion—by what has been called hydruria or polyuria, the urine at the same time frequently becoming albuminous."² This refers, however, of course, to the section of the nerves *above* the kidney, while here the lesion, whatever it was, must have been below that organ.

Very little attention has, I think, been paid to the diagnosis of direct wounds of the ureter. Gustav Simon, in the work above referred to, mentions no case in which the ureter was directly wounded, nor does he seem to contemplate the possibility of such an injury. In fact, if we consider the great depth at which the tube lies, its small size, and its close proximity to the peritoneum, we can easily understand that an uncomplicated wound of the ureter must be among the rarest of surgical injuries. Such, however, I believe, to have been the lesion in the case before us, and though I am sensible that there is always something unsatisfactory in the record of a case, the diagnosis of which is not absolutely certain, yet I thought the one here narrated, whatever view may be taken of it, was too extraordinary to be allowed to remain unrecorded. I hope that, by making it public, I may elicit some information from others, which may clear up its obscurities, or that at any rate it may be of use hereafter when some parallel case may occur, in which the nature of the injury can be verified anatomically.

Note.—Mr. Morris has been so kind as to send me the

¹ 'Textbook of Physiology,' p. 279.

² Hermann also gives similar though vaguer statements, 'Phys., translated by Gamgee,' pp. 115, 117.

following conjectural explanation of the peculiar nature of the discharge in this case, assuming that the ureter was really wounded.

“May it not have been that the ureter was a dilated one, from some congenital obstruction which had later on become removed, yet not before the kidney had been so far impaired as no longer to be able to separate the usual amount of solids? Such is the case in hydronephrosis, as I mentioned in a paper published in the ‘Med.-Chir. Trans.’ of last year, pages 253 and 254. This supposition explains both the liability to injury of the ureter and the characters of the urine, while we know that dilated ureter from congenital, as well as from non-congenital causes, is far from uncommon.”

This explanation is certainly very ingenious, and is to my mind a probable one, and I have noticed that a similar idea was propounded in a discussion on the case, which was published in a foreign journal, to which, however, I have mislaid the reference. I must leave the matter to the reader’s judgment.

I have not discussed the possibility that the fluid might have come from the peritoneal cavity. The *immediate* escape of a large quantity of fluid, and the absence of albumen, seem to me conclusive against any such explanation of the above case.

NOTE ON THE DELHI BOIL.

(FURUNCULUS DELHINUS.)

BY

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(Received March 12th—Read April 24th, 1877.)

THERE being readily accessible more than one good description of the above-named affection, my later observations need but a few introductory remarks.

1. That the “Delhi sore” or “boil” is a peculiar complaint is averred by the common testimony of those who by local experience have become acquainted with its manifestations, and such testimony is, at least, *prima facie* evidence of the reality of this complaint.

2. The main point, therefore, concerns its true nature, and on this question opinions differ. Thus, some medical men, practically conversant with the Delhi boil and its nearer congeners, have supposed that we see here a local complaint, due to infection of some kind from the soil or water-supply; whilst others, not so conversant and being led by general reasoning or analogy, have refused to admit the specific characters of the sore, or at most would refer the complaint to a syphilitic origin; and, finally, an opinion held by both classes of observers is this, namely, that the affection under notice is but a local sign of general deterioration of health. Though I have not

myself visited Delhi, yet I have seen instances of the "boil," and had occasion to peruse its ample though scattered records, besides having studied on the spot two similar cognate affections, and on comparing all these data, I have come to the following conclusion:—That whilst there is here certainly a peculiar skin-complaint, its identity with any form of syphilis cannot be maintained; and, further, the hypothesis of an antecedent cachexia is frequently both needless and insufficient. Hence, there remains to consider the facts pointing to specificity of character, and such are these:—Endemicity and a direct convectability—features in themselves well-nigh decisive, especially when conjoined with the asserted protectiveness of the malady against its own recurrence. Add, too, a certain periodicity of occurrence of outbreak (*e. g.* the month of March, 1876; the autumn is also named); a selection of the most exposed or accessible surfaces of the body (most evident in the better clothed European); a somewhat definite course and duration of the boil; its convectability to a distance; its local characters so marked as to warrant accurate popular diagnosis; and, finally, the comparative harmlessness of the whole thing, with its utter intractability to other than destructive treatment. It is also significant that new-comers or dwellers of not more than a year are often, and all ages and both sexes equally, liable to attack—frequently a whole body of individuals being affected in rapid succession, after the manner of a localised epidemic. I have intimated that bad health is no necessary concomitant of the "boil;" and as indicating a special rather than general influence, would include such negative features as singular exemptions, irregular onset, and variations in degree.

3. In brief, the balance of proof seems clearly in favour of the local origin of the Delhi boil; and as regards its faculty of spreading by contagion, there are so many modifying conditions—both personal and social—that most, if not all, exceptional instances in its dissemination

appear to be applicable upon this datum, at least as readily as upon other and less well-defined hypotheses. As at present informed, then, we should be warranted in maintaining that the early "papule" is due to irritation of some poisonous or parasitic agency, derived from without and settling on the part; that its enlargement into a scaly "nodule" or "boil" is consequent upon a local multiplication of such agent, and, with the "sore" or ulcerated surface which commonly but not invariably follows, would be anticipated from the attendant inflammation and degeneration of tissue. Perhaps, too, an "open" surface is favorable to the extrusion and fructification-products, and the serpiginous character of the ulcer is certainly significant. Discharges—dry or moist—seem to be a means of natural dissemination; auto-inoculation is likely, and a certain degree of systemic infection may follow, whereby arise a multiplication of "boils" and a final protected state of the constitution. There are facts on record intimating that the lymphatic system is sometimes visibly implicated; and, in conclusion, we see here perfectly definite characteristics and many seeming irregularities, to be so readily understood on the view of specificity that it would not be surprising to find direct evidence of foreign and peculiar agency within the structures which are the seat of the boil. What has been observed is the following.

4. By the kindness of Dr. Fairweather, civil surgeon at Delhi, I procured last spring several specimens of this local affection, which were taken from the living subject or excised shortly after death, being then preserved in diluted alcohol and solution of potassic bichromate. On arrival in Bombay, the parts were hardened in strong spirit, converted into thin sections, and submitted to microscopic examination with the aid of simple reagents, as glycerine, acetic acid, and solution of potass. Different stages of the sore were represented, but not all specimens were suited for minute scrutiny, owing to variations in the strength of the preservative media. Amongst those

in better condition there was one, measuring an inch across, of oval form, and moderate duration, representing the stage just prior to ulceration, which Dr. Fairweather described as "a beautiful specimen cut out entire, along with a margin of sound skin, from the cheek of a lad who was killed by lightning, and whose body was sent for medico-legal examination." My chief results were obtained from this characteristic sample, but the other specimens, stated to be also typical, furnished useful material; and I may briefly sum up the observations made as follows:—In the early stage of the boil there is some overgrowth of the epidermis and a defined cellular infiltration of the derma, encroaching also upon the subjacent connective tissue; afterwards the cuticle becomes not only thickened, but subject to irregular development. I noticed in it a group of large ($\frac{1}{1000}$ inch), dark ovoid cells filled with bright granules, which, however striking, might have been only sebaceous products, and there were seen collections of clear reniform bodies, possibly accidental; more noticeable, however, were the numerous globular masses of laminated epithelium, sometimes large enough to be visible to the unaided eye, generally of opaque-white hue, and situated both superficially and deep; a few darker "globes" were found to contain coiled-up hairs. I note that the hair-follicles were widened, varicose, plugged with opaque matter, and accompanied by the spherical masses just named; the hair-shafts are broken, clubbed, swollen, or shrunk, and of granular aspect; the sebaceous glands distended or withered, sometimes expanded into cysts, and their contents variously altered. The sweat-glands and ducts did not claim especial attention; no strictly abnormal elements were observed in these epidermal structures. The papillæ of the cutis were, prior to obliteration, greatly enlarged, often dipping far down and sometimes branched at their free ends; they and the rest of the true skin were the seat of a dense cellular infiltration, whose characters did not seem peculiar; it was the same kind of

granulation-tissue which I had found in the "bouton de Crete" and the "bouton de Biskra;" and I may add that the epidermal formations are mainly identical in "Caneotica" and the "Delhi boil." Blood-vessels were plentiful, and at first nothing more unusual was noted, but guided by previous experience the search was continued, and then I detected in the particular specimen already adverted to, the appearances which are depicted in the drawings. Of these, figs. 1, 2, 3, and 4, Pl. XIV, indicate the presence in dilated canals (probably lymphatic vessels) of a mycelium-like growth, which if not common had yet a characteristic aspect; it was not affected by the commoner reagents, is extremely minute, and of far too regular a form to be due to accident; certainly it came not from outside, but had arisen *in situ*. Figs 5 and 6 show an even more striking stage of the growth, and after due consideration I am disposed to rely upon the validity of these observations, as demonstrating the existence of a true parasitic plant-like structure within the substance of the Delhi boil. The aspect, position, relations, and disposition, and the uniform character of the filamentous mass last described cannot, in my opinion, be relegated to known structures, and to conceive of their artificial production seems impossible. Here but little granular matter was intermixed with the growth; elsewhere such was more common, and it is within the bounds of supposition that it may be of micrococcus nature. I have no desire to speculate upon the connection of these foreign materials with the essential cause of the Delhi boil, having no other data to offer, yet one may imagine that such connection exists, if it be not a vital one; and my observations seem to afford a definite basis for the view, otherwise supported, that the troublesome skin-affection under notice is really of parasitic origin, *i. e.* of specific character. They confirm, for me, the opinion that such an affection exists as was lately termed *mycosis cutis chronica* (*vide* the last volume of the 'Transactions' of this Society).

In conclusion, it is legitimate to add that light is hereby afforded in practical dealing with these peculiar endemic skin complaints; and future observers will now have a definiteness of aim, which may be useful for their guidance.

DESCRIPTION OF PLATE XIV.

(Delhi Boil, Dr. H. V. Carter.)

Fig. 1. In a section from outer part of sore: + K-O + glycerine: \times 500 diameters. Not far from the free surface, and near the edge, and surrounded by a dense infiltration of granular cells, is a pear-shaped cavity, containing a number of exceedingly minute and clear fibres, which have the aspect of branching filaments; they probably extend throughout this space. are mostly straight, colourless, and united at sharp angles. Transverse diameter of cavity, about $\frac{1}{200}$ in.; of filaments, probably less than $\frac{1}{20000}$ in. Another empty space is seen below and to the left; towards the right hand is part of a sebaceous gland. The cavity has defined and somewhat thickened walls, and intermingled with the filaments is a certain amount of fine granular matter. Nothing like *conidia* are seen, or free cells or nuclei. The dark tint below is probably due to red blood-material.

Fig. 2. Section from near middle of sore, at a spot not far from free surface: + acetic acid and glycerine: \times about 500 diameters. The open space displays a closer network of delicate, straight, and branching, bright fibres; refracting granules are intermingled, and, above, a clouded mass of granular cells, in which some of the fibres are implanted. Towards the narrower lumen an endothelial lining is observable. At *a* are fibres, seen with a power of 700 diameters, which appear as if tubular, and occasionally jointed or beaded. At *b* are two cells from the interior of the cavity, within which were noticed one or two bright short rods, resembling Bacillar bodies, such as I have observed in other places, *e.g.* in perfectly fresh (living) cells and juice from a leprous nodule, to which no reagent was added. My notes add that the above fibres are not due to interspaces of endothelium, not branches of adenoid cells, not yellow elastic fibres, and cannot be nerve-tubules. The structure is a real one.

Fig. 3. A fragment from the edge of another section: + acetic acid + glycerine: $\times 500$. Here is a small obliquely divided canal, which, besides a granule-cell or two and a small group of red granules, contains a similar network of fine fibres. These were seen also before the addition of the diluted acetic acid.

Fig. 4. From a section of the sore to which K-O and glycerine were added. It is an oblique vertical section, and the spot shown is not far from the surface, towards one end: $\times 700$. An elongated channel appears, which is formed by the bifurcation of a canal coming up from below. Both canal and branches are filled with a fine branching growth, having delicate filaments of bright aspect, which assume the characters of mycelium. A few bright granules are mingled, and the mass, in different foci, presents the appearance of a densely felted collection. The prolonged action of K-O renders the filaments less visible.

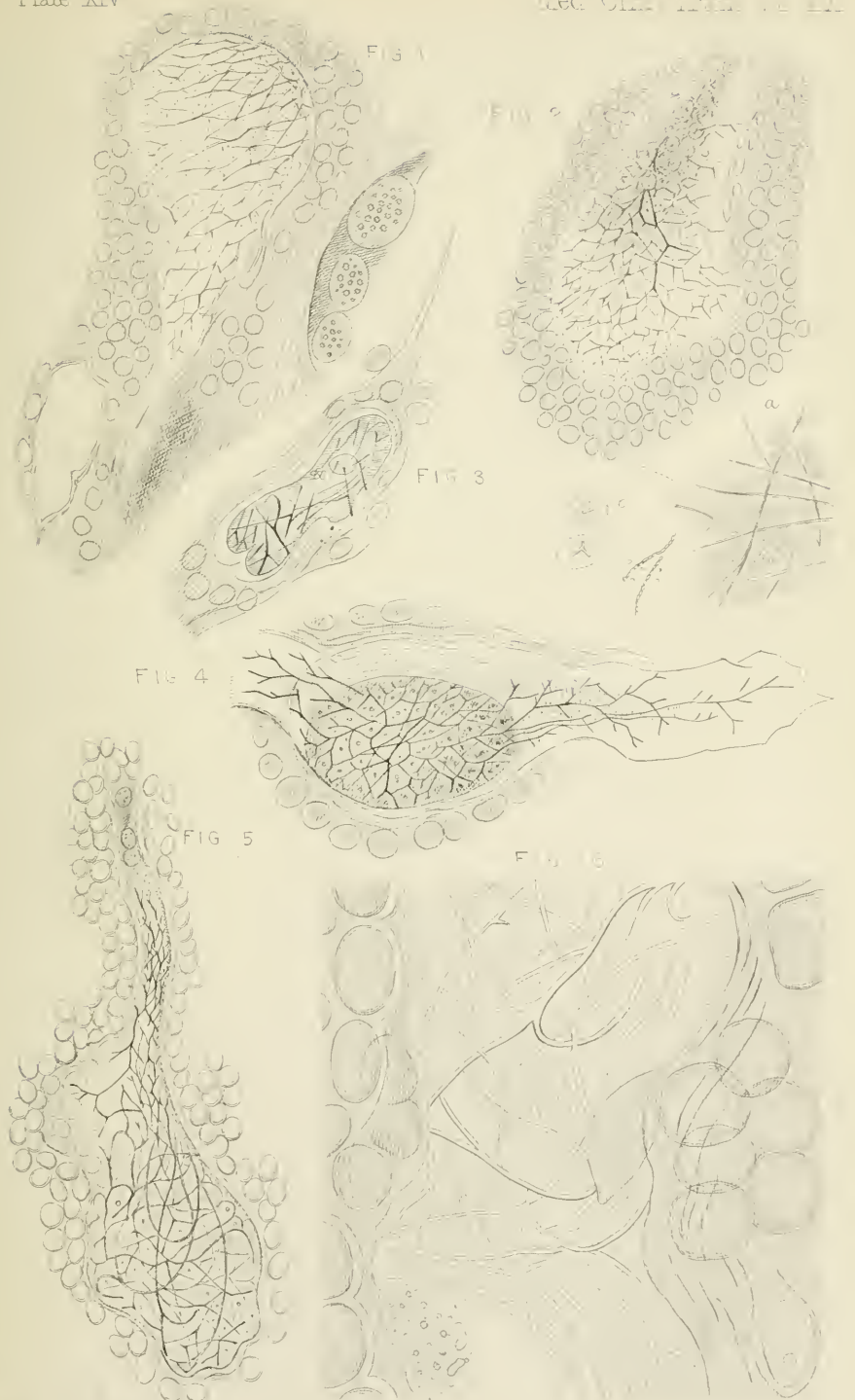
Fig. 5. From a section to which glycerine alone is added. At a thin edge is noticed the long vessel here depicted, which, in its sinuous course, rather abruptly expands into an elongated cavity, where is seen a mycelioid growth. This growth is very luxuriant, presents an angular aspect where most compressed, (?) in the narrower lumen, and more open or wavy meshes in the dilated portion; its mode of branching is clearly shown, and all is sufficiently defined. Nothing of the like aspect is visible outside. The unaltered vessel (? lymphatic) is just equal to the diameter of the granular cells it serves to convey, or $\frac{1}{3} - \frac{1}{4000}$ in. The dilated cavity measures $\frac{1}{500}$. The filaments are probably not more than $\frac{1}{20000}$ in., and they do not vary much in diameter, being remarkably uniform, and, to all appearance, *sui generis*.

Fig. 6. A part of the above, opposite the mark *; $\times \frac{1}{24}$ immersion, and about $\frac{1}{1800}$ diameters. On each side the walls of the canal are seen in varying focus. The interior is occupied by the new growth alone, whose filaments seem neither jointed nor beaded, are close set, and arranged as shown, it being impossible to depict their ramifications compatibly with clearness. Towards the wider end (above) the filaments are less densely aggregated, whilst in the direction of the narrow portion (below) they are more closely arranged and more parallel; here some granular matter exists which contains round, ovoid, and beaded, refractile bodies.

Sections from the scar of a Delhi boil all showed orange or red tinted particles, not unlike those found in the scar of the Bouton de Biskra, as well as remains of cell-infiltration. The above-described filamentous substance was not seen, nor, I may add, will it be easily detected in most sections of whatever age or stage of the 'boil.' In

the drawings the filaments are rendered by dark lines ; they have, in nature, however, a bright centre, due to refraction, which I am unable to represent.

Date of the above observations, June and July, 1876.



A C A S E
OF ALL BUT
UNIVERSAL PARALYSIS IN A CHILD,
FOLLOWING EXPOSURE TO HEAT;
WITH COMPLETE RECOVERY.

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Received March 27th—Read April 24th, 1877.

CATHERINE DIGRAINE, æt. $2\frac{1}{2}$ years, was brought to St. Bartholomew's Hospital on the 28th of July, 1876. She lay helpless and incapable of movement in her mother's arms. She was a small pallid child, with fair hair, brown eyes; was somewhat rickety, but fairly nourished.

On admission the face was a little flushed. The pupils were large and equal. Tongue clean and red. She was quite conscious and could speak as well as most children of her age. Respiratory movement was mainly abdominal, jerky and irregular, about 56 per minute. The

chest was everywhere resonant, and the breath-sounds weak. The position of the heart was normal, the sounds clear, and the pulse 156 per minute, regular and compressible. The abdomen was natural, save that the liver was palpable for one inch below the ribs on the right side. Axillary temperature 102.2° F.

It was observed that she had complete loss of motor power both in her legs and arms. She could move the left scapula, but not the right, could not raise her head from the pillow or support it when held up, but rotation from side to side was possible in the recumbent posture.

Observations as to the sensory condition were rendered difficult because of her age, and her habit of answering "yes" to almost every question; but there appeared to be anæsthesia of all parts affected with motor paralysis. She sometimes cried when one of the toes was pinched hard.

The muscles of all the limbs were extremely soft, wasted, and flabby. In the legs a strong Faradaic electrical current excited only slight reflex movements, but no muscles responded directly to this stimulus in any of the extremities.¹

The child lay on her back and passed both fæces and urine involuntarily, the bowels being frequently moved. No urine could be saved for examination. There was troublesome spasmodic cough, especially in the morning. The appetite was good, and there was no sickness.

The history of her case as elicited from her mother, after repeated cross-examination at different times, was as follows:—She was born in America, of Irish parents, and was always a healthy child till her present illness began. While travelling by rail to New York in very hot weather on the 23rd of June (1876), the child complained of severe headache and was very sick. The same night she had pains all over her, was sick, delirious, and very

¹ It was unfortunate that no constant-current battery was at hand at this time, and thus no information could be obtained as to the effect of continuous or interrupted Voltaism.

thirsty. The parents remained in New York for a week, and during this time the child got better. She had no loss of power and walked about.

On July 1st, while walking on the quay before embarking, during a very hot day, the child fell on her knees and suddenly lost all power over her limbs. There was no loss of consciousness. The same evening she was delirious and had no control of the sphincters. On the voyage homewards she slept a great deal, was frequently delirious, and took very little food. She had diarrhoea, but was not sea-sick. Her temper was irritable, and she used bad language.

The daily notes of the case after admission, including the conditions of the pulse, respiration, and temperature, carefully drawn up by Mr. W. S. A. Griffiths, the clinical clerk, are appended. From these are condensed the following particulars of the progress of the case. Light diet, with milk and arrowroot, was given at first, and half a drachm each of cod liver oil and syrup of phosphate of iron was ordered thrice a day. Some meat was soon after added to the diet.

In five days it was noted that no change had taken place in the child's condition. No sickness and no delirium occurred. A strong Faradaic current produced only slight muscular contractions in the arms, none in the legs. The motions and urine were passed involuntarily, but some of the latter was secured and found to be void of albumen.

On the 2nd of August Dr. Gee saw the case in consultation, and confirmed all the conditions previously noted. Upon his suggestion, belladonna was prescribed in doses of one third of a grain of the extract, in the form of a sweetened mixture thrice a day, and a strip of belladonna plaster was placed down the whole length of the spine. The steel and cod-liver oil were continued, and two ounces of port wine were added to the dietary. Faradization to the limbs was practised daily.

On the following day the tongue was clean and moist,

the pupils somewhat dilated, and the cough was more frequent. The respiration and pulse were quickened, and the temperature had risen from 99.2° to 102° . The urine contained abundance of lithates. The chest and abdomen were covered with a scarlet erythematous rash which faded on pressure. The same night the temperature rose to 103.6° . Next day the pupils were widely dilated, the rash had extended to the neck and thighs, and was fading on the chest and abdomen. A lumbricus, seven inches long, was passed *per anum*.

On the fourth day of the belladonna treatment it was noted that she could move the left hand a little. She took nourishment and slept well. The rash extended to the head, and some redness appeared over the sacrum. Her mother stated that the child had previously passed round worms; several doses of santonine were therefore given, but no more came away.

The belladonna treatment was now administered to the extent of one and one third of a grain in the twenty-four hours.

At 10 p.m. on this night—the fourth of the belladonna treatment—the temperature reached the highest point recorded during her illness, 104.5° .

Two days subsequently some dulness was noted at the bases of the lungs with moist sounds and rhonchus, but the temperature fell to 101.3° .

Next day a marked increase of power was noted in the left arm, the child being able to raise it to her mouth and to feed herself; there was also increased sensibility in the left thigh.

In a few days there was commencing power in the right arm and leg, but there was no control as yet over the sphincters. The temperature rose about one degree each evening, and reached 101° .

On the 12th of August, sixteen days after admission, the child could raise both hands over her head and feed herself; there was increasing power in the right leg, and sensibility in both legs. The pupils continued fully

influenced by the belladonna, but the tongue never became dry. The temperature varied from 99.4° to 100.6 .

On the twentieth day she was first able to control both sphincters.

A powerful Faradaic current only caused very slight muscular contraction in the limbs, and on the twenty-fourth day stronger response was noted, equal in the arms, but more marked in the right leg.

After being employed for twenty-one days, and on the twenty-fifth day from admission, the belladonna was omitted.

Little further improvement was observed in the course of the following week, but on being taken out of bed it was found that she could put one foot in front of the other when held¹ up. When lying down she was unable to crawl.

On the 29th of August the report was that her pulse was 156 (it had varied from 120 to 160 during the previous five weeks), respirations 44, and temperature 100.6° . The urine was natural.

A drachm of the syrup of phosphate of iron and lime, with one minim of liquor strychniæ, was ordered thrice daily.

On the 5th of September she was not so well. Her face was very pale and slightly puffy. The urine was "smoky," and contained one tenth of albumen, also granular casts and blood-discs. The pulse was 76 and irregular, respirations 28, and the temperature was observed to be normal for the first time. The tongue was clean and the appetite satisfactory. The cardiac and respiratory sounds were clear. Sensibility was improved in the arms.

In two days the pulse was more irregular, and the amount of albumen in the urine was larger. No pyrexia. Some sickness occurred at times.

On the 14th of September, ten days after the albuminuria was noted, there was only a trace of albumen in the

urine, and the child could stand erect with a little assistance.

A fortnight later Mr. Power examined her eyes with the ophthalmoscope, but found nothing abnormal.

On the 10th of October, seventy-five days after admission, she could stand and walk by herself round her bed. The urine was free from albumen.

Ten days later she could walk about the ward, in ten days more she was able to run, and in three months from the date of admission, and four months from the onset of the illness, she was in good general health, and enjoying perfect use of all her limbs and sphincters.

This case presents several features of interest and importance, which may be more particularly expressed under the heads of diagnosis, prognosis, and treatment.

Firstly, as respects diagnosis, it seems certain that the symptoms betokened an affection of the spinal marrow and muscular system, and not one involving the brain in any marked degree.

It may be stated at once that the case was at first regarded as an example of essential infantile paralysis, and for the following reasons:—The age and previous health of the patient, the character of the early symptoms, and the specific condition of the affected muscles.

It was, however, not easy to reconcile with this view of the case the sensory paralysis and the implication of the sphincter muscles.

The cerebral symptoms at the onset were as little marked as they commonly are in cases of essential paralysis. There were no convulsions or loss of consciousness, but there were delirium, sickness, and irritability of temper. The rule is that sensation remains unaffected in this form of paralysis;¹ in this case there

¹ "Sensation is almost never disturbed," Volkmann, 'German Clinical Lectures,' 1st series, New Syd. Soc. Translation. "Sensory disturbances are always absent," W. H. Erb, 'Ziemssen's Cyclopædia,' English Translation, vol. xi, p. 446.

was decided impairment, if not complete loss in some parts, of sensibility in all the limbs during the first few weeks.

Implication of the sphincters has, so far as we can learn, never been observed in these cases.¹

The condition of the muscles in respect of Faradaic electrical stimulation was precisely that which is met with in cases of true essential paralysis of children after the second week of the disorder.

It is perhaps not too much to assert that the whole subject of infantile paralysis is as yet somewhat obscure, and requires further investigation. The following classification probably includes most forms of disease included under this name.

First, there is that form which may be described as Kennedy's paralysis.² In these cases the symptoms are observed at a very early period of life, and are for a short time severe. One or more limbs may be affected. Gastric or gastro-enteric troubles appear to be the exciting cause, and complete recovery is the rule within a few days or weeks.

Secondly, a form of infantile paralysis has been described which is due to exposure to cold, and is sometimes called rheumatic.³ Thus, an extremity, usually one of the lower limbs, is affected, the paralysis bearing perhaps no relation to any particular nerve or set of nerves. Muscular atrophy follows. In this form there is probably some trophic change in the substance of the muscles, and it is found that a slowly-interrupted Voltaic current shows signs of increased irritability in them, which Faradization hardly, if at all, evokes.

Thirdly, there is a class of cases occurring in young children, as well as in adults, where the mischief is

¹ "The bladder and rectum are never involved," Volkmann, *loc. cit.*

² Henry Kennedy, M.B., 'Dublin Medical Press, Sept., 1841; 'Dublin Quarterly Journal, Feb., 1850, and Nov., 1861.

³ 'Clinical Uses of Electricity,' J. Russell Reynolds, M.D., F.R.S., p. 42, London, 1871.

limited to one limb usually, and results from mechanical injury to the principal nerves. This paralysis may be termed truly peripheral. Violent pulling or dragging may cause it, and the symptoms betoken the effects of a local nervous concussion. Thus, pain with paralysis of motion and sensation ensue, also wasting of the limb.

Recovery commonly follows, but the cases may be tedious, and the extremity may never reach full development.

Cases such as these have been described by Sir James Paget.¹

Fourthly, there is the largest class of cases which includes the gravest and most unpromising features of infantile paralysis. In this the early symptoms are usually of sudden onset in children of the age of two or three years, and who have generally been otherwise robust. One or more extremities are found to be paralysed. The early history commonly throws no light upon the nature of the case. There may be only a day or a night of previous poorliness; but in a certain number of these cases there is at the outset a very painful condition of the affected members, and so much local disturbance as to bring the cases under surgical rather than medical observation. This painful phase, which may simulate deep-seated inflammatory trouble, soon passes off, and leaves the limbs in the ordinary limp condition. Such cases very rarely terminate in perfect recovery; generally one limb remains more or less affected for life.

The case described above does not seem naturally to fall into any of the classes just enumerated. There appears, further, to be no relation between any of these classes of infantile paralysis. Such knowledge as is possessed respecting the early pathology of the fourth or gravest class of infantile paralysis, points to a form of myelitis specially affecting the anterior horns of the grey matter, and the anterior and lateral columns of the spinal

¹ "Clinical Lecture on some Cases of Local Paralysis," 'Med. Times and Gazette,' vol. i, 1864, p. 331.

cord, and thus it appears almost too much to look for perfect recovery of all functions after so serious and extensive a lesion. Hence it seems fair to conclude that a less severe affection was present in our case. The special treatment by belladonna was certainly adopted in view of some such condition of the cord, and its results were not unnaturally held at the time to be sufficiently satisfactory, and as favouring the opinion held by therapeutists that this drug has an influence in checking spinal hyperæmia.

The fact of ultimate *perfect* recovery in our case might in itself throw grave doubt upon a diagnosis of true infantile paralysis. Volkmann and others of large experience in these cases declare that perfect recovery is hardly known, and it is at least possible that some of the alleged recoveries may have been in cases of peripheral or rheumatic paralysis.

Failing to establish an unequivocal diagnosis of infantile spinal paralysis in this case, it became necessary to seek some other explanation of the symptoms. It was not possible to attribute the paralysis to any distinct strumous affection, or to any nervous disease specially related to, or specifically dependent upon, the amount of rickets which was present in the child. Neither could the existence of lumbrici in the intestines be regarded as a cause, for no such symptoms are recognised amongst the reflex nervous phenomena due to these parasites. Moreover, the child had been subject to worms before her illness, and certainly no decided improvement took place in her case upon the expulsion of that one observed while she was in hospital.

We therefore came next to the conclusion that the probable etiological explanation of the case was to be found in the fact of direct exposure to the sun and severe thermal influences. The history was clear that the child had been exhausted, badly tended, and exposed to the sun in New York (which in the month of July is well known to be a particularly hot city), before she sailed for England.

The strongest argument that can be fairly adduced against this view of the case is, that there is no experience on record, so far as we are aware, of such nervous symptoms due to insolation. Such are, however, quite conceivable, and they may possibly occur and yet not be plainly recognised or traced to their true cause.

The condition of the muscles must not, however, be quite left out of view. It seems not impossible that the same thermic influences which induce disorder in nervous structures may also involve the muscular system independently, and with especial severity in the case of a frail and ill-nourished child. We are therefore disposed to regard this extensive paralysis as due to profound nervous exhaustion, the muscles being, perhaps, simultaneously affected, and thus the affection would seem to fall into the category of so-called functional rather than of distinctly organic disorders. The spinal marrow, and not, as occurs most commonly, the brain, suffered.

The lesions, whatever they may have been, were clearly recoverable ones.

The occurrence of albuminuria at the beginning of the third month, lasting as it did for five weeks, is not perhaps quite easy of explanation. This symptom is not met with either in cases of infantile paralysis or of insolation, and, so far as it goes, determines equally against either diagnosis. The presence of an erythematous rash at one period of the case, followed in some weeks by albuminuria, might not unnaturally suggest the possibility of an intercurrent attack of scarlet fever. But it is to be noted that the rash appeared immediately upon the employment of full doses of belladonna and its application to the spine, and no sore throat, desquamation, or other symptoms of scarlatina were observed. There were also no cases of that disease in that wing of the hospital at the time.

The albuminuria is not, therefore, to be thus explained. It seems not improbable that this symptom admits of the same interpretation as the other more prominent

phenomena of the case, viz. profound nervous exhaustion.

Prognosis.—Under this head there is little to be remarked. The case looked very unlikely to recover till some days had elapsed from its admission to the hospital, and for some weeks but small hope was entertained of even partial recovery of one or more of the extremities.

Upon the recognition of a similar condition, a hopeful prognosis might naturally follow, supposing that a favorable climate with supporting treatment and wholesome hygienic influences could be secured.

Treatment.—Respecting this, it may be briefly stated that the general debility and the condition of the child plainly indicated good nourishment and wine. Steel and cod-liver seemed called for.

During the progress of the case the temperature-curve followed a course in no special degree characteristic.

On admission the thermometer registered 102.2° , and at the beginning of the treatment by belladonna it was 99.2° . On the second day of this medication it rose to 102° , and an evening rise of about one degree occurred for the greater part of the time any pyrexia lasted.

The first symptoms of amendment were observed on the fourth day after taking belladonna, and gradual improvement ensued both in recovery of sensory and motor functions. The muscles were regularly stimulated with powerful Faradaic currents for several weeks till active response took place.

Strychnia was given in small doses at a late period.

We cannot doubt that the proper early treatment for a case of this nature should consist of cold affusion, as in the more ordinary forms of heat-stroke, and it seems not unlikely that by this means paralytic sequelæ might be either reduced in severity or altogether averted.

The following are the daily notes of the case:—

Catherine D—, æt. $2\frac{1}{2}$, admitted July 28th, 1876.

Her mother brought her from Liverpool on the 28th, where they had just arrived from New York.

July 30th.—She is a small, pale, rickety child, fairly nourished, light brown hair and eyes. Pupils rather dilated, equal, face slightly flushed. Tongue clean, rather red. She is quite conscious, and can speak as well as other children of her age. Pulse regular, compressible, 156. Respirations, chiefly abdominal, irregular, jerky, 56. Temperature 102·2°.

Thorax.—Thoracic walls move but little with respiration. Respiratory murmur everywhere feeble, otherwise normal. Percussion note resonant everywhere.

Heart.—Position normal; action rapid; no bruit audible.

Abdomen.—The liver can be felt one inch below margin of ribs in the right hypochondrium. Abdomen otherwise normal.

She has complete loss of power in both legs and arms. She can move the left scapula, not the right. She cannot raise her head from the pillow, or hold it up when it is raised, but she can rotate it when lying down.

Owing to her age and her answering “yes” to almost every question, her power of sensation is doubtful, but there appears to be complete anæsthesia of all the paralysed parts. She sometimes cries when one of her toes is firmly pinched; as a rule she appears quite unconscious of pain.

She lies on her back in bed, able to rotate her head, but not to make any other movement. Motions and urine passed involuntarily.

The muscles of her arms and legs are very soft and flabby. Very slight reflex action is produced in the legs by a strong Faradaic current; none in the arms.

She has a troublesome spasmodic cough, especially in the morning. No sickness. She takes food well. Bowels loose. Urine cannot be saved for examination.

She was born in America, of Irish parents, and had been a healthy child until the present illness. The

parents were coming home to England, and on June 23rd, 1876, whilst travelling by train to New York, in very hot weather, the child complained of severe pain in the head and was very sick. Towards night she complained of pains all over her, was very feverish, thirsty, and became delirious. They remained a week at New York, and she got better. During that week she had no loss of power and could walk.

On July 1st, a very hot day, whilst walking on the quay to the ship about midday she fell on her knees, and from that moment without losing consciousness she lost all power in her limbs and control over her sphincters. At night she became delirious, and, according to her mother, throughout the voyage she took very little food, slept much, was frequently delirious, and when so used very bad language. She did not suffer from sea-sickness, but constantly from diarrhœa. At the end of the voyage she had made no improvement.

Since admission she has not been delirious, and her mother thinks she is more lively than she was a week ago. Half diet, with arrowroot.

℞ Syrupi Ferri Phosphatis;
Olei Morrhuæ, āā ʒss. Fiat mistura.
Ter die sumenda.

August 1st.—No improvement in power of movement. Strong Faradaic current produces only slight muscular contractions in the arms, none in the legs. She takes her food well. Motions and urine passed involuntarily. Urine free from albumen. Less cough. Pulse, fair volume, compressible, 132. Respiration 60. Temperature 99·2°.

2nd.—In much the same state. Wine ʒij.

℞ Extracti Belladonnæ, gr. ⅓;
Syrupi Aurantii, ʒss;
Aquæ menthæ piperitæ, ʒj.
Fiat haustus, ter die sumendus.

Faradization to the limbs daily. Emplastrum Belladonnæ 2 by 12 to the spine.

3rd.—Pupils rather dilated. Cough more troublesome. Tongue clean and moist. She takes her food well. The chest and abdomen are covered with a red erythematous rash, disappearing on pressure. Pulse, same characters, 148. Respiration 64. Temperature $102\cdot0^{\circ}$.

10 p.m.—Urine loaded with urates, no albumen. Pulse 156. Respiration 48. Temperature $103\cdot6^{\circ}$.

4th.—Pupils fully dilated. Passed a good night. Tongue clean and moist. The rash has extended to the thighs and neck, fading on the chest and abdomen. Pulse 160. Respiration 40. Temperature $101\cdot6^{\circ}$.

10 p.m.—This morning she passed a round worm (seven inches in length). Pulse 144. Respiration 44. Temperature $102\cdot4^{\circ}$.

5th.—She continues in the same state. Takes food fairly. Sleeps well. Pupils rather dilated. Bowels act two or three times a day. She frowns a good deal. The rash has extended to the face and almost disappeared from the body. Some redness about the sacrum. She can move the left hand a little at the wrist. Pulse regular, 120. Respiration 60. Temperature $102\cdot5^{\circ}$.

10 p.m.—Pulse 156. Respiration 48. Temperature $104\cdot5^{\circ}$.

Repetatur Hst. Belladonnæ, 6tis horis;

R Santonini, gr. ij.

Hæc nocte sumend. et rep. cras mane.

7th.—Slept badly last night. She is more fretful. Cough more troublesome. Takes her food well. Pupils still dilated. Rash almost gone. No increase of power. Redness about the sacrum is increased. Resonance impaired at base of both lungs. Breathing harsh. Scattered moist sound and rhonchus. To have a water cushion. Pulse 136. Respiration 60. Temperature $102\cdot3^{\circ}$.

10 p.m.—Pulse 152. Respiration 36. Temperature $101\cdot3^{\circ}$.

8th.—Slept fairly well. Still very fretful. Takes food

well. Bowels open. Motions and urine passed involuntarily. Tongue clean and moist. There is a marked increase of power in the left arm and hand; she can raise it to her mouth and feed herself. Increase of sensation in left thigh. Urine loaded with lithates, no albumen. Pulse regular, compressible, 144. Respiration 40. Temperature $100\cdot6^{\circ}$.

10 p.m.—Pulse 144. Respiration 36. Temperature $100\cdot0^{\circ}$.

9th.—Rather more power in right hand. No more worms passed. Her mother says she has passed worms frequently before this illness. Pulse 120. Respiration 36. Temperature $104\cdot0^{\circ}$.

10 p.m.—Pulse 156. Temperature $101\cdot0^{\circ}$.

10th.—Better night. Less fretful. Motions and urine still passed involuntarily. Pulse 136. Respiration 36. Temperature $100\cdot5^{\circ}$.

10 p.m.—Pulse 150. Temperature $101\cdot3^{\circ}$.

11th.—There is great improvement in right arm. Some in right leg. Pulse regular, 132. Respiration 40. Temperature $100\cdot0^{\circ}$.

12th.—She can raise both hands over her head, and can use the fingers of both hands to scratch it. She can feed herself and can hold her head up. Sphincters still do not act. Power in right leg increasing. Sensation slowly returning in both legs. Pulse regular, compressible, 140. Respiration 40. Temperature $99\cdot9^{\circ}$.

10 p.m.—Pt. Emp. Belladonnæ. Temperature $100\cdot1^{\circ}$.

13th.—More cheerful. Sleeps well. Bowels open. Pulse 120. Temperature $99\cdot4^{\circ}$.

10 p.m.—Temperature $100\cdot6^{\circ}$.

14th.—Condition same. Pulse 150. Temperature $99\cdot9^{\circ}$.

10 p.m.—Temperature $100\cdot4^{\circ}$.

15th.—Motions and urine still passed involuntarily. General condition improved. Pupils dilated. Tongue clean and moist. Pulse regular, 132. Respiration 40. Temperature $100\cdot4^{\circ}$.

16th.—Pupils still dilated. Less cough. Fretful.

Pulse small, regular, 132. Respiration 40. Temperature 99·6°.

18th.—Yesterday and to-day she has shown signs of returning power and sensation about the sphincters, telling nurse when the bowels are about to act and when she wishes to pass water. Increased power in both legs. Feet kept extended. Very slight muscular contraction produced by very strong Faradaic current. Pulse compressible, regular, 132. Temperature 100·0°.

19th.—Continues to improve. Temperature 98·8°.

21st.—Sleeps well. General health improving. Power over sphincters gradually returning. Sensation generally improved. Muscles contract more with Faradization, arms equally. Right leg more than left. Urine 1018, acid, no albumen. Pulse 100. Temperature 98·7°.

R. Olei Morrhuæ, ʒj;
Syr. Ferri Phos., ʒss. Fiat mistura.
Ter die sum.

22nd.—Continues improving. Omit Hst. Belladonnæ.

28th.—Cough troublesome. Sleeps well. Appetite good. Bowels open. Pulse 140. Respiration 40. Temperature 101·0°.

29th.—Improvement less marked. Usually calls for stool. When supported can put one foot in front of the other. She is not able to crawl on the floor. Urine normal. Pulse small, 156. Respiration 44. Temperature 100·6°.

R. Syr. Ferri Phos., ʒj;
Liq. Strychniæ, ʒj. Fiat mistura.
Ter die sum.

September 5th.—She is not so well. Face very pale, slightly puffy. Appetite good. Bowels open. Tongue clean and moist. Heart sounds clear. Respiratory murmur clear. Improvement has been less marked during the past week. Arm more sensitive to Faradization. Urine smoky, contains a small quantity of albu-

men (about one tenth) with granular casts and a few blood discs. Omit Liq. Strychniæ. Pulse soft, irregular, 76. Respiration 28. Temperature 98·4°.

7th.—She did not sleep well last night. Takes food well. Tongue moist, thin white fur. No diarrhœa or sickness. Urine contains more albumen. Pulse more irregular, 80. Temperature 98·3°.

8th.—She was sick last night, but looks better. Regaining power in her limbs. Urine contains less albumen. Pulse irregular, 88. Temperature 99·3°.

12th.—She is better. Takes food well. Bowels open, action usually voluntary. Has not vomited since the 10th. Tongue clean and moist. Urine contains less albumen, excess of urates. Omit Ferr. Phos. Pulse soft, irregular, 134. Respiration 40. Temperature 99·4°.

℞ Tinct. Ferri Perchlor., $\text{m}\nu$;
Glycerini, mxx ;
Aquæ ad ʒss . Fiat haustus.
Ter die sumendus.

14th.—Improving. Less pale and anæmic. She can stand with a little support under the shoulders. Urine contains a trace of albumen only. Pulse regular, soft, 108. Temperature 99·2°.

18th.—More power in arms and legs. Tongue clean and moist. Pulse regular, compressible, 120. Temperature 97·6°.

19th.—Urine still contains a trace of albumen with granular casts, much granular débris, and a few red blood discs. Pulse 120. Temperature 99·0°.

26th.—She continues improving rapidly. Complexion less pasty. Slight cough. Can sit up without support. Sensation of legs still much impaired. Urine slightly clouded with albumen. Pulse small, regular, 132. Temperature 99·7°.

28th.—An ophthalmoscopic examination was made to-day by Mr. Power, no intra-ocular change was found. Pulse regular, 140. Temperature 98·6°.

October 3rd.—She continues improving in both sensa-

tion and motion. Bowels open. Appetite good. Tongue moist, thin white fur. Urine contains a trace of albumen. An egg. Pulse small, slightly irregular, 126. Respiration 22. Temperature 97.1° .

10th.—Improving. General health good. Can stand and walk round a bed by herself. Urine free from albumen. Pulse 124. Respiration 30. Temperature 98.6° .

17th.—She can walk across the ward with a little help. Pupils less dilated. Urine free from albumen. Pulse small, regular, 140. Temperature 99.2° .

20th.—Can walk across the ward by herself. Pulse regular, 106. Temperature 98° .

25th.—Continues improving. Urine, no albumen. Pulse compressible, 100. Temperature 98° .

31st.—She can run down the ward. General health good. Pulse, fair volume, 100. Temperature 98° .

November 5th.—Discharged well. Can run up and down stairs. Sphincters quite under control. Muscles have not regained their proper tone, but are still soft and flabby, though much less so than they were on admission.

ON AN
INTERNATIONAL CALCULATING SCALE
FOR OPHTHALMOLOGICAL PURPOSES.

BY
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(Received April 10th—Read April 24th, 1877.)

ALTHOUGH the metrical system of numbering lenses has been unanimously adopted by the Congress of Ophthalmologists of 1875, and the International Medical Congress of the same year, I believe it is yet the exception to find an English oculist prescribing, or an English optician supplying, glasses according to that measurement. Believing, however, that this system has certain advantages, and that since it has been adopted by other European nations we shall follow them, I have caused the scale to be made, which I beg to bring before the Society, as a means of facilitating the introduction of the system into England.

It consists of six calculating tables, viz. the metrical system (Dioptrics), the French system, the German system, and the English system of numbering lenses, and two tables giving the presbyopia and the accommodation present at certain ages.

M. Javal, of Paris, was, I believe, the first to adapt the

system of calculation, by means of a sliding rule, to the purposes of ophthalmology. He, twelve years ago, had a scale made with the numbers corresponding to the old French system engraved on it, so that the usual additions and subtractions of that system could be made. This scale has been enlarged, so as to meet the requirements of the metrical system, and the German, English, and metrical scales calculated according to their respective measurements. We have thus not only the calculations of fractions in any one scale, but also at a glance a comparison of any one system with any of the other systems, and the resolving of the product of any calculation into the system required. (See diagram.)¹

The following calculations can be rapidly made.

The *addition* of the fractions or integers of the four systems is made as follows. The arrow of the sliding scale corresponding to the system in which the number is calculated, is placed opposite to the smaller fraction or integer, and the figure opposite to which the larger fraction stands in the same system is the product of the calculation. For example, if it is desired to add $\frac{1}{18}$ th to $\frac{1}{9}$ th, the arrow on the sliding scale being placed opposite to the 18 on the fixed scale, 9 on the sliding scale will be found opposite to 6, which is the answer.

The *subtraction* is made by placing the smaller fraction on the sliding scale opposite to the larger fraction in any one system, and noting to which figure the arrow stands opposed. For example, to subtract $\frac{1}{15}$ th from $\frac{1}{3}$ rd, the 15 on the English sliding scale being placed opposite to 3 on the English fixed scale, the arrow will be found to stand against $3\frac{3}{4}$, which is the remainder.

Any power given in any of the systems can be added to, or subtracted from, any of the other systems and the

¹ The diagrams (Figs. 1 and 2) represent the two sides of the scale; they are the actual size in breadth, but only about a third of its length, the original measure extending to above 20 on the dioptric scale, and being eighteen inches in length.

| | | |
|---|--|-----------|
| D | 0.25 0.50 0.75 1 1.25 1.50 1.75 2 2.25 2.50 2.75 3 3.25 3.50 3.75 4 4.50 5 5.50 6 6.50 7 8 | DIOPTRICS |
| P | 0.25 0.50 0.75 1 1.25 1.50 1.75 2 2.25 2.50 2.75 3 3.25 3.50 3.75 4 4.50 5 5.50 6 6.50 7 8 | FRENCH |
| F | 0.25 0.50 0.75 1 1.25 1.50 1.75 2 2.25 2.50 2.75 3 3.25 3.50 3.75 4 4.50 5 5.50 6 6.50 7 8 | FRENCH |
| D | 0.25 0.50 0.75 1 1.25 1.50 1.75 2 2.25 2.50 2.75 3 3.25 3.50 3.75 4 4.50 5 5.50 6 6.50 7 8 | DIOPTRICS |
| G | 0.25 0.50 0.75 1 1.25 1.50 1.75 2 2.25 2.50 2.75 3 3.25 3.50 3.75 4 4.50 5 5.50 6 6.50 7 8 | GERMAN |
| G | 0.25 0.50 0.75 1 1.25 1.50 1.75 2 2.25 2.50 2.75 3 3.25 3.50 3.75 4 4.50 5 5.50 6 6.50 7 8 | GERMAN |
| D | 0.25 0.50 0.75 1 1.25 1.50 1.75 2 2.25 2.50 2.75 3 3.25 3.50 3.75 4 4.50 5 5.50 6 6.50 7 8 | DIOPTRICS |

FIG. 1.

| | | |
|------------------|--|---------------|
| D | 0.25 0.50 0.75 1 1.25 1.50 1.75 2 2.25 2.50 2.75 3 3.25 3.50 3.75 4 4.50 5 5.50 6 6.50 7 8 | DIOPTRICS |
| E | 0.25 0.50 0.75 1 1.25 1.50 1.75 2 2.25 2.50 2.75 3 3.25 3.50 3.75 4 4.50 5 5.50 6 6.50 7 8 | ENGLISH |
| E | 0.25 0.50 0.75 1 1.25 1.50 1.75 2 2.25 2.50 2.75 3 3.25 3.50 3.75 4 4.50 5 5.50 6 6.50 7 8 | ENGLISH |
| D | 0.25 0.50 0.75 1 1.25 1.50 1.75 2 2.25 2.50 2.75 3 3.25 3.50 3.75 4 4.50 5 5.50 6 6.50 7 8 | DIOPTRICS |
| E | 0.25 0.50 0.75 1 1.25 1.50 1.75 2 2.25 2.50 2.75 3 3.25 3.50 3.75 4 4.50 5 5.50 6 6.50 7 8 | ENGLISH |
| P _W | 0.25 0.50 0.75 1 1.25 1.50 1.75 2 2.25 2.50 2.75 3 3.25 3.50 3.75 4 4.50 5 5.50 6 6.50 7 8 | PRESBYOPIA |
| A 75 70 65 60 55 | 45 50 55 60 65 70 75 80 85 90 95 100 105 110 115 120 125 130 135 140 145 150 155 160 165 170 175 180 185 190 195 200 | ACCOMMODATION |

FIG. 2.

product reduced to any system required. Thus, a French myopic patient may bring to an English oculist a French minus lens of $\frac{1}{7}$ th, which suits him for distance, and asks for a weaker lens on the metrical system to use at a given distance, say eighteen inches. By placing the 18 on the sliding scale of the French system opposite to 7 on the fixed scale of the same system, the arrow will be found opposite 3.25 of the metrical system, which is the glass he requires to place his punctum remotum at eighteen inches. Or, should an English oculist wish to give a patient with a hypermetropia of $\frac{1}{10}$ th a glass to suit his presbyopia of fifty years according to the metrical system, the arrow of the English sliding scale is placed opposite to the 40 of the presbyopic table, the age at which presbyopia is supposed to commence, and the amount corresponding to the age of fifty, viz. 2 dioptrics read off. The same arrow is then placed opposite to 10, the amount of the hypermetropia on the English fixed scale, and the 2 on the dioptric sliding scale will be found nearly opposite to 6 of the dioptric fixed scale, which is the number required to suit his hypermetropia and presbyopia combined.

Calculations combining the tables for presbyopia and accommodation, which have been calculated so as to correspond with the recent tables of Donder's, with any of the other tables given, can be quickly made. The examples given above will sufficiently explain this.

Any one already in possession of a set of lenses can see at a glance the number of the metrical or other system to which any lens corresponds, and may thus prescribe lenses according to the metrical system, though using trial lenses corresponding to any other system.

The ophthalmoscope accompanying the scale, which was shown at the meeting and which I described elsewhere in 1873, has had metrical lenses introduced, instead of those of the English system. By using single glasses thirty powers are at command, while by combining the glasses over one hundred different powers are obtained, ranging

from 18 to $\frac{1}{4}$. The same points of an ophthalmoscope are preserved, which I formerly considered necessary, viz. a small mirror which allows of a near approach to the eye; a large aperture of about $3\frac{1}{2}$ millimètres in the mirror to prevent any stenopæic action; the aperture having a knife edge and being carefully bevelled off so as to prevent any sign of a tunnel, which is apt to cause a reflex and limits the field of vision; the close apposition of the lens to the aperture for the same purpose; an easy revolution of the discs containing the lenses, but not by means of the finger on the periphery, which prevents the observer going close to the eye; and fair sized lenses in the discs.

TWENTY-FIVE CASES OF OVARIOTOMY,

WITH SOME

REMARKS ON THE CAUSES AND TREATMENT OF THE
FEVER SO FREQUENTLY FOLLOWING THE
OPERATION.

BY

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(Received April 5th—Read May 8th, 1877.)

IN presenting a record of twenty-five cases of ovariotomy to this Society so soon after Mr. Spencer Wells has enriched our experience by a paper on 300 cases, I should feel some apology to be necessary did I not believe that every addition to our knowledge of this important operation will be welcome to the profession.

To Mr. Wells' kindness I am indebted for many of the cases in the accompanying table, and to the opportunities I have enjoyed of assisting him in the greater number of the cases he lately brought before us is due very much of the knowledge which has enabled me to attain a fair amount of success.

Though dealing with a total of cases so relatively small as mine, it is still impossible even to refer to the points of special interest which the individual cases bring before us. I propose, therefore, to direct the attention of the Society

to the causes of the high temperature which so frequently follows the operation, and to the methods of treatment found most useful in lowering it.

Fever, or high fever, may arise after ovariectomy from several causes, two of the most important being septic changes in the blood and serum effused into the peritoneum immediately after the closing of the abdominal incision, and peritonitis resulting from the exposure and handling of the peritoneal surfaces, or their contamination with blood or ovarian fluid. Peritonitis, if divorced from its connection with the septic condition, is, I believe, much less common than we might expect, less common, indeed, than is generally supposed.

There is, however, I believe, another form of fever frequently becoming quickly high fever, distinct from the two named above. When speaking of fever I mean any condition in which the temperature is over 100° F. and under 103° F. When the latter degree is exceeded I shall call it high fever.

I will now endeavour to define this third kind of fever and then offer, what seems to me, a natural explanation of it, in the hope that a criticism of my views by those Fellows of the Society whose knowledge of the various feverish states is wider and deeper than my own may lead to an extension of our information on this subject.

Frequently after ovariectomy the patient's temperature rises rapidly and steadily, from a few hours after the operation, without this rise of temperature being associated with any of the symptoms which we regard as evidence of the presence of peritonitis or septicæmia—without, in fact, our being able to explain its presence by any recognised pathological condition. Or a similar state arises a few days after the operation, the patient meanwhile having perceptibly suffered more or less from a feverish condition, indicated more by the tense full pulse, flushed face, and urine loaded with urates, than by any very high temperature.

If the temperature continue high, or continue to rise beyond the point I have defined as my fever limit, we

soon have, as the result of it, lesions of important organs, to which our attention is directed often, I think, to the exclusion of their original starting-point. I do not know that I have ever seen a case in which the fever was fatal, because some result of it is sure to step in and hasten the final catastrophe. In other cases, again, nature seems equal to the task of restoring the normal condition, and we have nothing left to show the danger from which our patient has escaped.

So much for what, I fear, is a very imperfect explanation of what I mean when I speak of the fever following ovariectomy. How, then, are we to explain this condition? If we look to our case-books we shall see that this state is much more common after simple uncomplicated operations performed on young or healthy women than after those performed when the ovarian disease has already lowered the vitality of the patient, or when extensive adhesions have led to some loss of blood and a prolonged and severe operation. In this, I think, we have the key to the position. An ovarian tumour is a highly vascular growth, and from its position and surroundings there is little to interfere with its blood-supply. After the tumour has been exposed by the abdominal incision we tap it and empty it more or less completely. Its walls, besides being highly vascular, are very elastic, and though the arteries are provided with unusually thick muscular coats, which give them considerable power of resisting any impediment to their circulation, the veins are often large and have thin walls, hence, when the tumour is empty, or nearly so, and its walls contracted, the calibre of the veins is much reduced, and they oppose the free circulation of the blood. The arteries, already resisting compression, are not equal to this double opposition, and the tumour becomes comparatively bloodless. The more simple the cyst the more markedly is this the case, and but little blood is removed with it after its pedicle is secured. The patient is then, after the operation, in much the same condition as a patient after the operation of transfusion.

She has not really more blood, *always a little* less, but it has a *much* smaller area to circulate in. The arteries and veins of the general circulatory system are fuller of blood, and the tension is increased. The heart has harder work to do ; it acts more forcibly and more rapidly, the blood is circulated more quickly, and the tissue changes are proportionately more rapid. The result is a considerable rise of temperature. The same condition may often be seen after tapping an ovarian cyst, though in a less degree, as we might expect, for the cyst is not emptied of all its blood, nor is the circulation cut off.

Turn, now, to the various organs likely to be affected by this increased rapidity of the circulation with an increased volume of blood. The brain, we know, receives a relatively large portion of the blood circulating through the body. The nerve-centres, then, are stimulated with the other organs or to a greater degree. In some cases the stimulus is not greater than necessary for a response to the greater activity throughout the tissues ; in others it is too great, and we have an improper or irregular action of the nerves of nutrition added to the already high-pressure action in the economy. The balance is destroyed, and we have developed in some organ, less able than the others to bear the strain, a pathological state which we recognise and which is apt to take our attention from the primary cause. We think that its diseased state was the cause of the high temperature and other symptoms which have been causing us some anxiety. It may be the brain itself shows symptoms of giving way, and if this is the case grave, indeed, is the danger ; the lungs, the heart, or the kidneys may be the organs affected. Our treatment, then, in any case should be directed to the control of the circulation and the lowering of the temperature, and we should do this from the earliest symptom of a tendency to steady rise of temperature, or some one important organ may have already suffered, though as yet it has given us no indication of its approaching failure.

If I have made my meaning sufficiently plain it will be

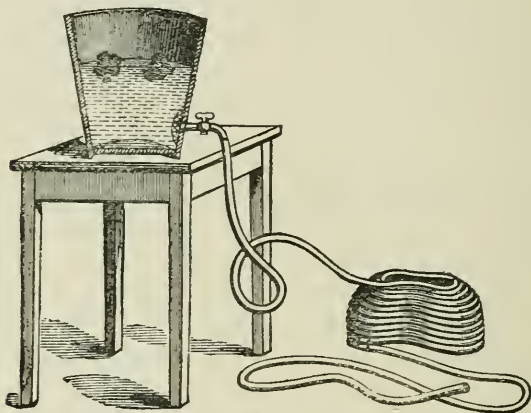
at once seen that, in order to control the circulation and lower the temperature, we must apply our treatment to the most important organ which is concerned, with all the others, in the danger. Fortunately, the brain, which is the most important, both from its large blood supply, and also from its regulating power over the whole system, is also the most easy of access for suitable treatment. If we can keep it cool and regulate its blood supply we may hope that the other organs, with the aid of a healthy nerve-action, will in time overcome the difficulties in which they are placed.

The ice-water cap which we now commonly use at the Samaritan Hospital, and which, I am told, is coming extensively into use on the Continent and America, admirably meets all the requirements of our case. I am not prepared to define exactly its mode of action, *i. e.* whether it is by the general cooling of the whole volume of blood or by its direct action in keeping the nerve-centres cool, and cooling their more immediate blood-supply. I think, when we consider the large quantity of blood passing through the brain, we may fairly suppose that the ice-water cap has some general action in lowering the temperature of the whole blood, but from carefully watching many cases in which it has been used, I am inclined to think that the direct action on the nerve-centres is its most important one. Possibly it may lessen the quantity circulating by causing contraction of the arteries, thus both reducing the volume and cooling it at the same time.

After an ordinary meeting of this Society, held April 27th, 1875, when my colleague, Dr. Day, read a paper on "Chest Complications of Abdominal Tumours," I had the honour, as a visitor, of showing one of these caps, and I afterwards published a short account of it, with an engraving, in the 'Medical Times and Gazette,' May 27th, 1876. I have now placed one of the caps on the table for inspection, and a copy of the engraving named above accompanies this paper (see woodcut, p. 302).

As at present used it was made for me by Messrs.

Krohne and Sesemann, being a modification of one which I had found very useful while I was house-surgeon to Professor Lister, in Edinburgh. The original cap was the invention of a working man connected with some india-rubber works in Glasgow.



The pieces of ice shown in the water are much too small; one large piece answers best.

Before proceeding to point out the advantages which I think it possesses over all other methods in use for reducing temperature, especially in the cases under present consideration, I think it may be of interest to the Society if I briefly trace the history of the use of cold for reducing temperature after ovariectomy. The condition which I have attempted to describe and explain had been noted by Mr. Wells before I was acquainted with his practice and various methods of applying cold had been tried, among them Roberts' pads, Richardson's ice-water collar, and the more common bladder of ice to the head, and cold sponging.

I believe the first introduction of coiled tubing, with a constant stream of water passing through it, is due to Dr. Roberts, of Manchester, who published an interesting paper on this subject in the 'Medical Times and Gazette,'

December 16th, 1871, the same year in which Dr. Wilson Fox read his paper before the British Medical Association on the use of the cold baths in the hyperpyrexia sometimes occurring in rheumatic fever. Dr. Roberts' cases show a much slower fall of temperature than is usual with the ice-cap, his plan being to lay the patient on a pad of coiled tubing, or wrap it round the body. The effect on the pulse and respiration is also, in some of his cases, *nil*, in others but slight, and this is especially worthy of notice in connection with the question of general blood cooling or action on the nerve-centres. He also found the temperature rise to a higher degree than ever after the pad was withdrawn; I have never seen any such results when using the cap.

Dr. W. B. Richardson suggested the employment of a collar of coiled tubing, so as to cool the blood in its passage to the brain. Mr. Wells had used this before I was acquainted with his practice, and we tried it again at the time we were first using the ice-cap. Before trying it, I theoretically objected to it on the ground of the small circulating area it covered, the area being also traversed by large vessels, and the blood passing in considerable volume and rapidly across it. This objection was found sound in practice, for but little effect was produced on the temperature by its use, and it was also found troublesome to keep in place and irksome to the patient.

The first copy of my old Edinburgh friend fully answered my expectations, but from having merely four large chambers partially shut off from one another it was troublesome to manage and often out of order. Once it actually burst while in use, fortunately, without causing any injury to the patient, except some little alarm. While we were thus still not satisfied with our means of treating the pyrexia Dr. Broadbent kindly undertook to pack one of Mr. Wells's cases in the wet sheet on the principle found so successful in fever cases. The result in lowering the temperature was very remarkable, and the patient was undoubtedly rendered much more comfortable,

and had her life prolonged for some days, but the labour, both for Dr. Broadbent, who kindly came frequently to superintend the packing, and for the nurses was great, and the constant moving of the patient very undesirable in a surgical case. The same objection applies to the cold bath found so useful by Dr. Wilson Fox and others in in treating hyperpyrexia in medical cases.

The cap, as at present used, is made of tubing, with one flat side, round tubing being a failure, as the air between the coils became warm. The advantages it possesses over other methods of lowering the temperature are its easy application (the size of the patient's head being all the measurement necessary in ordering one), and its giving a steady, dry, cold application, which does not require any movement on the part of the patient when it is applied, and does not tire or weaken her. Frequently the patient falls into her first tranquil sleep since the operation shortly after it is put on, and seldom is any objection raised to it; often a desire is expressed that it may be kept on after the surgeon thinks it no longer necessary. The cap when applied is connected with a pail of water raised a foot or so above the patient's head, a large block of ice being placed with the water in the pail, the other end of the pipe which, coiled, forms the cap, passes into a pan at the bedside, and the water, as it runs through, may be returned to the pail. At the hospital we now have a special supply cistern, with tap at proper level, in each ward.

How far its direct action on the nerve-centres also gives it an advantage over and above the other methods of lowering temperature, I must leave to future experience to decide.

In my own practice I order the nurse always to apply it if the temperature passes 102° , and I frequently order it on with a much lower temperature, if the pulse is hard and full, or if there is flushing, with headache or other cerebral symptoms. Once used I prefer to keep it on till the temperature falls to normal, but generally give both nurse

and patient some latitude, leaving it off for a time, then on again, as the constant filling of the pail is hard work.

In thus giving the first place to the ice-water cap I must not, however, neglect other remedies, which, I think, may in some cases be useful; I refer to blood-letting and aconite.

I think, in some cases, venesection is undoubtedly very useful, as it relieves the heart and circulation generally of the very thing which I have endeavoured to show is the cause of all the mischief, but to be useful it must be employed very early and tolerably freely, and I am inclined to think that with the dry cold, applied efficiently and early, it might be altogether dispensed with. I think aconite, given in very small doses (half a minim every half hour), acts beneficially by steadying the heart's action and lessening the tension of the pulse, but I cannot say that I have ever seen it reduce temperature or have ever felt very sure that its use rendered the skin moist, though I know great authorities would be against me here. I think both venesection and aconite might probably be dispensed with whenever we can secure a full supply of ice and efficient means for applying it.

Having thus considered this form of fever and its treatment at some length, the question very naturally arises, How are we to distinguish it from high temperature due to peritonitis or some form of septicæmia? I am afraid in the majority of cases we cannot do so in the first instance, though we may make a shrewd guess in many cases, if we carefully note the expression of the patient, the comparative rate of the respiration and pulse, and other general symptoms. In the simple fever the patient is usually flushed, has bright eyes and tranquil expression, perhaps complaining of the heat, or of some giddiness, noise or pain in the head, but otherwise expressing herself as quite well. The respiration is seldom hurried, unless the lungs are beginning to suffer, when it may rise rapidly to an extent altogether uncommon in the early stages of septicæmia or peritonitis. The pulse is hard and full, and

incompressible, but often not unduly fast, at least, not so fast as in other conditions causing high temperature.

In both septicæmia and peritonitis the expression of the patient's face helps one much. In the former there is often a wild, startled look, which once seen is not readily forgotten, and the patient answers questions in hurried, jerking, manner; the pinched, anxious face of peritonitis needs no description here. I may remark that I dread a rapid pulse during the first forty-eight hours after ovariectomy more than a high temperature, especially if the former be small and hard, as distinguished from the full hard pulse I have been speaking of.

Of course, peritonitis or septicæmia will be met with as a complication of this simple fever, and in these cases we can only wait and watch the result of our treatment before deciding on what the high temperature depends. Fortunately, the ice-water cap is beneficial in all these fevers, though the same cannot be said of venesection and aconite. Venesection, while it may be useful in peritonitis, I believe to be dangerous in any septic condition, and I do not think the aconite does any good in peritonitis, while I believe it to be dangerous in septicæmia as tending to increase the depression.

I will now endeavour to illustrate this theory from some of the cases recorded in the table which is before the Society, though there is not among them one typical case, because, having had some considerable experience in after-treatment from Mr. Wells' cases before I operated on cases of my own, I always go upon the good old rule of prevention being better than cure, and apply the ice-cap always if the temperature gets up to 102° , and frequently if it reaches 101° with a tendency to rise, and the other symptoms of the fever are present.

Case 1 affords an illustration of those in which the fever rapidly seizes upon the weak point in some important organ and calls away our attention from itself. At the time I operated in this case I did not know the value of the ice-water cap, and I allowed my attention to be

directed to the lungs, imagining that I had an ordinary case of acute bronchitis to deal with. The operation was one of the most simple I have ever seen. For twenty hours the patient seemed to be doing well, though she was flushed, had a full, hard pulse, 120, temp. 101° , and resp. 20. She suddenly began to complain of difficulty of breathing, and in two hours had a pulse of 140 and resp. 32, with dry sounds all over the chest. I treated her as a case of acute bronchitis till the fourth day, when the chest symptoms disappeared, but the temperature was 103.4° and steadily rising. I then bled her from the arm to twelve ounces with some relief, but again the temperature rose and her other symptoms were aggravated; I tried the wet pack and then again bled her, and after eight days of great anxiety she died. I feel sure that had I used the ice-water cap in this case the symptoms would have given me little trouble. Its fatal termination made a deep impression upon my mind, partly, doubtless, from its being my first ovariectomy, but still more because I felt that such cases ought not to die, and that we needed some more efficient treatment for them. I afterwards had the advantage of treating a similar case of Mr. Wells' at the Samaritan, in which the temperature on the morning of the second day after operation was 100.6° , pulse 100, respiration 20; three hours later I saw her with temperature 103.4° , pulse 120, respiration 32, and an exactly similar chest condition to that of my own patient. The ice-water cap was immediately put on; before I left her the breathing was easier, and in a few hours the temperature was falling steadily; the pulse was 112, and the respiration 24, and all chest symptoms had disappeared.

In Case 6, seven hours after the operation, the temperature was 101.6° , pulse 132, respiration 30, with the other fever symptoms; no improvement had taken place after five hours' use of aconite, the cap was put on, and immediate and permanent improvement took place.

In Case 14 there was an unsatisfactory state for forty-

eight hours, but though constantly hesitating whether to begin the cap, I did not do so. The temperature then mounted to 102.8° , and it was at once put on; in the first nine hours afterwards the temperature fell two degrees, in the next three hours another degree, with a more than proportionate improvement in pulse and respiration and general symptoms.

In Case 16 the pulse was, from the first, irregular, full, thrilling, and incompressible. The patient, a young, full-blooded girl. When the temperature reached 101° , eight hours after the operation, the cap was put on; the temperature began to fall at once, and though the pulse remained fast, 120 for twenty-four hours, it then settled down, and the patient made a good recovery, not without my having some suspicion, from the action of the kidneys, that we had had a narrow escape of some serious mischief in them during the short time the pyrexia remained unchecked.

I have not had among my own cases examples of nephritis with albuminuria, but I have seen several such, and in Case 14, already alluded to, there were evidences of kidney irritation, with passage of large quantities of almost colourless urine of very low specific gravity. I have seen one case, after simple tapping, in which the fever ran very high for some days, even though controlled by the ice-cap, and the urine was for some time loaded with albumen. The drawing I hand round was made from the sediment of her urine. She ultimately made a good recovery.

I might multiply examples of the benefit derived from the use of the cap, not only in these cases, but in undoubted cases of peritonitis and septicæmia, but my paper has already exceeded its intended limit, and I am anxious to give a brief analysis of the cases contained in the table.

Before concluding this part of my paper I must, however, state, that since we used the ice-water cap, I have never seen a case, starting as one of simple fever after ovariectomy, pass on to high fever, some other morbid

condition, and death, while I have seen more than one such before, besides my own recorded above.

It will be seen from the table that seven cases out of twenty-five terminated fatally, a mortality of 28 per cent.

One of these, Case 5, would more properly find a place under fatal cases of tapping during pregnancy, for the patient was semi-comatose and moribund when I operated, but rallied afterwards sufficiently to talk to her husband, and lived sixteen hours—longer by several hours than was possible without the operation. This case is fully described in the twenty-seventh volume of the 'Transactions of the Pathological Society,' so I will not refer to it further here.

Cases 2 and 4 were almost hopeless from the reduced state of the patients, with certainty of extensive adhesions, but tapping being no longer useful I thought it right, after explaining to them the great risk, to give them the last chance afforded by ovariectomy.

Cases 11 and 15 were both severe operations performed on not very promising subjects, but I have seen many worse recover well. Both were performed in the same nursing home, both passed through high fever to coma and death on the fifth day. In both cases the wounds healed well, and there was no abdominal distension.

Case 11 was an enormous dermoid tumour of right ovary, the solid part, as removed, weighing nineteen pounds; the other ovary was also removed.

Case 15 was kindly seen with me in consultation, by Sir James Paget, who, while expressing doubt, thought the case one of an irregular form of pyæmia.

Case 18, the other fatal case, was operated on at her own home, in a country town. She was an old and very fat woman, and had been tapped twenty-five years before in St. Bartholomew's Hospital by our present distinguished President and Mr. Stanley. It was also a dermoid cyst. The operation was interesting as being a pure enucleation, no ligatures being required. She died in thirty-seven hours of rapid septicæmia, and I afterwards discovered

that the back of the premises was occupied by a slaughter-house and candle manufactory.

Case 7 was as bad a one as it is possible to conceive; the tumour ruptured into the peritoneum the night before the operation, and was extensively adherent to bladder, uterus, and intestines. The operation occupied two hours, both ovaries being involved in the mass removed. The ice-cap was put on ten hours afterwards; temperature 103.6° ; pulse 134, very feeble; respiration 28. To its use and the method of drainage brought to the notice of the Society in Mr. Wells' paper, I believe she owed her recovery.

Drainage with the glass tube from the first was used in six cases, four of which died and two recovered.

In one case I inserted an india-rubber tube into the peritoneum at the bottom of the incision, about a week after the operation; she recovered.

I think Case 18 would have had a better chance with drainage from the first.

Cases 3, 7, 11, 13, 17, 19, and 21, were all double ovariectomies, a most unusual proportion.

It will be seen, also, that in the great majority of the cases extensive adhesions were encountered.

Cases 11, 17, 18, and 19, were all dermoid tumours, also an unusually large proportion; in 17 and 19 both ovaries contained dermoid structures, a condition I have only twice before seen in a large number of cases.

| No. | Medical attendant. | Place of operation. | Condition. | Date of operation. | Adhesions. | Length of incision. | Treatment of pedicle. | Weight of tumour. | Result. | Subsequent history or cause of death. |
|-----|----------------------------------|---------------------|---------------------|--------------------|---|---------------------|---------------------------------------|-------------------|---------------|---------------------------------------|
| 1 | Mr. J. K. Thornton..... | Hospital | Married | 1874 23 Nov. | None | Inches. 4 | Clamp | Pounds 25 | Died 8th day | Peritoneal hæ- matocele. |
| 2 | Mr. Wilcox, Aylesbury.. | Hospital | Single | 1875 44 Feb. | Parietal, hepatic, and pelvic | 6 | Clamp | 28 | Died 37 hours | Exhaustion ? |
| 3 | Dr. Hardman, Blackpool | Nursing home | Single | 32 Aug. | None. Both ovaries | 6 | Both ovaries Clamp and ligature | 9 | Recovered | Well Mar., 1877. |
| 4 | Dr. Thorburn, Man- chester | Hospital | Married | 29 Oct. | Parietal, omental, me- senteric, and hepatic cyst | 7 | Clamp | 27 | Died 31 hours | Exhaustion ? |
| 5 | Dr. Parsons, Wimbledon | Hospital | Married Pregnant | 28 Oct. | None. Gangrenous cyst | 4 | Ligature | 6 | Died 16 hours | Septic peritonitis. |
| 6 | Mr. Edgar Barker | Hospital | Married | 35 Dec. 1876 | Parietal | 5 | Clamp | 32 | Recovered | — |
| 7 | Dr. Percy Boulton | Hospital | Married | 38 April | Parietal, omental, in- testinal, vesical, and uterine | 5½ | Both ovaries Ligatures | 13 | Recovered | Well Mar., 1877. |
| 8 | Dr. Clement Godson ... | Hospital | Married | 45 May | Parietal and vesical... | 5 | Clamp | 28 | Recovered | Well Mar., 1877. |
| 9 | Dr. Ballard, Huntingdon | Hospital | Widow | 35 July | None. Ruptured cyst | 5 | Clamp | 20 | Recovered | — |
| 10 | Dr. C. Bramwell, Not- tingham | Hospital | Widow | 38 July | None | 5 | Ligature | 10 | Recovered | Well Mar., 1877. |
| 11 | Dr. Brice Smith, Belfast | Nursing home | Married | 26 Sept. | Parietal | 8 | Both ovaries Ligatures | 26 | Died 5th day | Congestion of brain. |
| 12 | Dr. Archer, Wardsworth | Hospital | Married | 39 Oct. | Omental and intestinal | 5 | Ligature | 3½ | Recovered | Well Mar., 1877. |
| 13 | Dr. Geo. Pearce, Leices- ter | Hospital | Single | 21 Nov. | None | 4 | Both ovaries Ligatures | 26 | Recovered | Well Mar., 1877. |

| Case | Medical attendant. | Place of operation. | Condition. | Age. | Date of operation. | Adhesions. | Length of incision. | Treatment of pedicle. | Weight of tumour. | Result. | Subsequent history or cause of death. |
|------|--|---------------------|------------|------|--------------------|---|---------------------|---------------------------|-------------------|---------------|---------------------------------------|
| 14 | Dr. Ferguson, Cheltenham | Hospital | Married | 47 | 1876 Nov. | Omental | 6 | Enucleation and ligature | Pounds 28 | Recovered | Well Mar., 1877. |
| 15 | Dr. Dove, Stowmarket. | Nursing home | Married | 50 | Dec. | Intestinal and pelvic | 5 | Enucleation and ligature | 16 | Died 5th day | Congestion of brain ? pyæmia. |
| 16 | Dr. Kidd | Hospital | Single | 19 | Dec. 1877 | Parietal and omental | 4 | Ligature | 22 | Recovered | Well Mar., 1877. |
| 17 | Dr. Lloyd, Llandilo..... | Hospital | Married | 37 | Jan. | Parietal, omental, and pelvic | 5 | Both ovaries Ligatures | 26 | Recovered | Well Mar., 1877. |
| 18 | Dr. Phillips, Bromley ... | Private house | Married | 56 | Jan. | General to capsule ... | 5 | Enucleation | 6 | Died 37 hours | Septicæmia. |
| 19 | Mr. Hall, Fleetwood ... | Private house | Single | 25 | Jan. | Parietal and omental | 5 | Both ovaries Ligatures | 18 | Recovered | Well Mar., 1877. |
| 20 | Dr. John Murray, Norton, Stockton-on-Tees | Hospital | Single | 18 | Jan. | Parietal and omental | 5 | Ligature | 38 | Recovered | Well Mar., 1877. |
| 21 | Mr. Stevens, Hoddesdon | Hospital | Single | 24 | Jan. | Intestinal | 4 | Both ovaries Ligatures | 9 | Recovered | Well Mar., 1877. |
| 22 | Mr. Green, Portsmouth. | Hospital | Married | 42 | Jan. | Parietal, omental, and intestinal | 4 | Ligature | 18 | Recovered | Well Mar., 1877. |
| 23 | Mr. Palm, Hull | Hospital | Widow | 40 | Feb. | Parietal, intestinal, pelvic, and uterine | 5 | Enucleation and ligatures | 31 | Recovered | Well Mar., 1877. |
| 24 | Dr. W. H. Day | Hospital | Single | 19 | Feb. | Parietal, omental, and intestinal | 5 | Ligature | 23 | Recovered | Well Mar., 1877. |
| 25 | Surgeon-Major Will and Dr. Strother, Sheerness | Hospital | Married | 32 | March | Parietal and omental | 5 | Ligature | 22 | Recovered | Well Mar., 1877. |

A CASE
OF
PRIMARY CYLINDRICAL EPITHELIOMA
OF THE LUNG,

WITH SECONDARY DEPOSITS IN THE PLEURA,
BRONCHIAL GLANDS, AND LIVER.

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PRIMARY CANCER of the lung in any form is a comparatively rare disease, and instances in which this particular variety has been found are so rare as to induce us to bring the following case before this Society. We shall consider it first of all from its clinical and then from its pathological point of view.

H. P—, by trade a ship's carpenter, æt. 37, was admitted into the Royal Hospital for Diseases of the Chest, City Road, under the care of Dr. Finlay, on February 8th, 1877.

Previous history.—He stated that he had enjoyed good health until about six months ago, when, after getting wet through, he had suffered from cough and shortness of breath, with pain in the left infra-axillary region which

was aggravated by deep inspiration. For the last three weeks he had not been much troubled with the pain, but his difficulty of breathing had steadily increased, and he had rapidly lost flesh. There was no history of family constitutional taint.

State on admission.—The patient is in bed and lies by preference on his back or right side; he is unable to lie on his left side on account of pain. His expression is anxious; face dusky; lips livid; *alæ nasi* dilating with each inspiration; external jugular veins prominent and distended; fingers somewhat clubbed, especially those of the left hand. On removing his shirt for the purpose of physical examination, he is noticed to be a thin spare man, and greatly emaciated. The chest is somewhat barrel-shaped, its movements being impaired, mostly so on the left side. The percussion note is over-resonant over both fronts where are heard also loud sonorous and sibilant râles with prolonged expiration. Similar sounds are heard over the right back, and in the left infra-axillary region well-marked coarse friction. In the left back there is absolute dulness on percussion over the lower two thirds, above which the dulness gradually diminishes. Over the dull area the breathing is feeble and is accompanied by sonorous râles and prolonged expiration; vocal vibration is diminished, but not abolished. The præcordial dulness is much diminished and the heart's impulse marked in the epigastrium. The abdomen seems natural; there is no oedema of feet or legs. He has a troublesome cough, accompanied by the expectoration of tenacious pink-coloured sputa. Pulse 120, full and bounding; respirations 20, laboured; temperature 99.4° ; bowels regular; appetite bad; urine scanty, sp. gr. 1030, acid, depositing lithates on cooling, not albuminous.

There was but little change in his physical condition during the next nine days (February 17th), and with the view to determine or exclude the presence of fluid, two exploratory punctures were made through the chest wall on the left side near to the inferior angle of the scapula.

Nothing was obtained beyond a few drops of a thick blood-stained fluid, which was found on microscopical examination to contain several large compound granule-cells. It was noticed that the inguinal glands on both sides were slightly enlarged and hard. His breathing continued laboured, but became paroxysmally worse at times, particularly in the night.

On the 23rd, although by this time it had become sufficiently clear that he was suffering from intra-thoracic disease of a malignant nature, the puncture was repeated at a different spot, under the impression that there was also some fluid present. About a drachm of semi-transparent blood-stained fluid was withdrawn by the exploratory syringe, but on using the aspirator only about an ounce of thick blood-stained fluid was obtained.

Three days later (the 26th) the percussion note was very much impaired over the front as well as back of the left lung; and in two days more (the 28th) it was absolutely dull, the dulness not extending further to the right than the left border of the sternum: the breath sounds were more feeble, and at the apex tubular in character. It was also noticed that the supra-clavicular glands on the right side were now enlarged and very hard, which materially strengthened the diagnosis of intra-thoracic malignant disease. The heart's maximum impulse continued in the epigastrium and did not seem to be affected by the increasing dulness to which reference has just been made.

After this there was no material change in the physical signs. He emaciated rapidly, his breathing became more distressed, and he died on the 7th of March, one month from the time of his admission.

His urine was frequently examined, and beyond a deposit of lithates, presented nothing abnormal.

His bowels were regular throughout.

His temperature presented no remarkable features. Its highest point was 100.6° ; its lowest 97.6° ; and the daily average about 99° .

Autopsy twenty hours after death.—Rigor mortis moderate. Body much emaciated. Supra-clavicular glands on right side enlarged and hard. On section the outer portion was white and somewhat glistening, the centre softened down into a puriform material.

The anterior mediastinal glands, four or five in number, each about the size of a hazel nut, were also enlarged and hard, and resembled on section those above described. Neither pleural cavity contained fluid. On the surface of the right parietal pleura were several nodules of a soft spongy nature, the largest not exceeding an inch in length by half an inch in breadth. They did not involve the adjacent bony or muscular structures. Elsewhere the pleura appeared natural except at the base, where the surfaces were roughened in places, and presented some recent adhesions.

The right lung was large and heavy, weighing three pounds; its surface studded all over with numerous more or less projecting nodules, underlying the visceral pleura varying in size from a pin's head to a hazel nut. Similar nodules were found scattered throughout the entire lung. The superficial portions were emphysematous. The bronchi exuded on pressure a frothy curd-like material, and their mucous membrane was injected and thickened.

The left pleura presented some adhesions at its outer part, and was so firmly adherent posteriorly that it was impossible to remove the lung without tearing its substance.

On the parietal surface were several nodules of the same character as those on the right side.

The left lung was also large. It weighed 2 lbs. 10 oz.; and the pleura covering it was roughened in places by exudation. It presented patches of emphysema, and its surface was covered with nodules similar to those on the right lung. At the upper part of the lower lobe was a large mass of soft (cancerous) material, projecting slightly from the surface, measuring in length about five inches, and in breadth about two. On section it presented a yellowish-

white pulpy appearance, was soft and putty-like in consistence, and irregularly stained with extravasated blood. It yielded on pressure abundant thick puriform fluid, and the parts of lung tissue around were soft and friable. On cutting into the lung it was found to be œdematous, and nodules similar to those on its surface were seen disseminated throughout its substance. These growths, too, were composed of the same spongy material already described, and readily broke down on pressure. The bronchi contained thick curdy fluid, and the mucous membrane was injected and thickened.

The posterior mediastinal glands, about six in number, were enlarged, especially those on the left side. The largest was the size of a small walnut and pressed on the left bronchus, but not so as to narrow its calibre to any extent. They presented the same characters as those before described.

The liver appeared natural on its surface, but on cutting into it three masses of the same cancerous material were found, each about the size of a small hazel nut, two in the right and one in the left lobe. The organ weighed 3 lbs. 14½ oz.

The right kidney presented on its surface two yellowish spots about the size of pins' heads.

Heart.—The right ventricle contained a firm, decolorised, apparently ante-mortem clot; the left also contained a quantity of clot. The substance of the organ was natural, as were also the valves, excepting the tricuspid, the segments of which were somewhat opaque and thickened.

The spleen appeared natural.

The intestines were examined as far as the sigmoid flexure, and presented no abnormal appearance.

There were a few enlarged glands about the cardiac end of the stomach, and some of the retro-peritoneal glands along the spine were also enlarged, and presented the same characters as those before described.

Microscopic examination.—The portions of the organs intended for microscopic examination were hardened in

spirit, and the sections stained with logwood and mounted in dammar varnish.

Lungs.—For the most part there is a well-defined line of separation between the lung tissue and the substance of the new growth ; but in places this separation is less well marked. Fig. 1 (Plate XV) shows the cancerous deposit in the midst of the lung structure, in the interior of the alveoli themselves, the alveolar walls being quite distinct ; they are, however, considerably thickened by infiltration of cells probably of an inflammatory origin. Fig. 2 (Plate XV) shows another arrangement ; here there is a thick wall between the lung and the cancerous new growth. In both places, however, the structure of the growth is identical. When examined with a higher power this growth is found to consist of an alveolated stroma (fig. 3) ; the alveoli are filled with cells of a very typical, columnar form ; they are huddled irregularly together in some places, while in others they are arranged with great regularity around the walls of the alveoli, which then assume the appearance of the crypts found in an ordinary polypus of the rectum. The alveoli vary considerable in size, in places they are about as large as an ordinary pulmonary alveolus, in others they are much larger, as though from coalescence of several alveoli. This, indeed, seems to be the manner in which the cancer has spread ; the alveolar wall being pressed upon by the invading new growth has finally yielded to the pressure and broken down. The parenchyma surrounding the tumours is undergoing changes which belong to the category of catarrhal inflammation, and which have led to the distension of the alveoli with a highly corpusculated infiltration. In many alveoli this has become caseous. The epithelium is fairly regular in shape and of a typical columnar variety ; the cells are arranged in single layers for the most part, but occasionally they are two or even three deep. In no place do they seem to be ciliated. We could not trace any direct communication between the bronchial tubes and any of the cancerous nodules.

Pleurae.—The parietal layer of the pleura was normal in the greater part of its extent, but here and there it was studded over with flat-topped nodules, which seemed to grow from its surface. On examining thin sections, however, the new growth was found to affect almost the entire thickness of the membrane. Fig. 5 (Pl. XVI) shows the appearance which it presented under a low power, and fig. 6 represents a portion of the same section more highly magnified. It will be seen that the cylindrical character of the epithelium is well developed, and that it accurately corresponds in structure with that found in the lungs. The pleura is much thickened and infiltrated with cylindrical epithelial cells, which in some places seem to lie without any definite arrangement, but which in others are placed around the walls of alveoli. In some of the alveoli the epithelial lining projects into their interior (as at Pl. XVI, fig. 6 A), resembling in some measure an intestinal villus. The alveoli vary from minute dots to the size of split peas; they are mostly circular, the larger ones being seemingly formed by coalescence of several smaller ones; they are all lined by columnar cells, which are sometimes one and sometimes two or three deep.

Bronchial glands.—The gland examined was about as large as a good-sized filbert. On cutting across it, it was found to consist of two parts; a caseous portion—probably the original gland—and a spongy part slightly pigmented. This latter portion is found to be composed of the same kind of epithelial growth as that already described in the lungs and pleura. Fig. 4 (Pl. XV) shows the appearances seen under the microscope.

Liver.—The growths in the liver, though presenting characters very similar to those already described, were less well characterised. Not only were the individual cells less typical in form, but they were also less definitely arranged in cylinders. The line of separation between the new growth and the liver-cells was less abrupt than in either lung or pleura. There was well-marked infiltration of

leucocytes along the immediate line of contact, and the liver-cells themselves were very obviously altered in consequence. At a short distance from the new growth the liver-cells and the parenchyma assumed their normal appearance.

Remarks.—Clinically, the interest in the case just narrated, lies in its diagnostic side; as in the present state of our knowledge, treatment, except such as is directed to the relief of distressing symptoms, is obviously hopeless. Several cases of primary cancer of the lung have been recorded, but most of these have been mistaken for chronic tubercular disease, and one was diagnosed as capillary bronchitis with pneumonia.

In the present case the physical signs pointed rather to a limited collection of fluid with thickened pleura. The absence of cachectic appearance (owing, no doubt, to the asphyxial tint of the skin), the well-marked pleuritic friction in the left lateral region which was noted on admission, and the fact of the heart's maximum impulse being seen and felt in the epigastrium, together with the history of the onset of the disease, all gave probability to this diagnosis.

But the rapid emaciation, the paroxysmal character of the dyspnœa, the nature of the sputa, the absence of œgophony, the fact that the bronchitic râles were well heard over the dull area, and that the vocal fremitus was not abolished, the enlarged and hard cervical glands, and last and chiefly the negative results of the exploratory punctures, pointed with overwhelming force to the true nature of the disease.

A case reported by Dr. (now Sir George) Burrows in the twenty-seventh volume of the 'Transactions of the Royal Medical and Chirurgical Society,' February 13th, 1844, the nature of which was diagnosed during life, presented many points of agreement with the present case; but no microscopical examination was recorded, and, indeed, this seems to have been generally, if not always, omitted.

Only two cases, so far as we can find, have been re-

corded hitherto, which the microscope has shown to be of a similar nature to the present case, and it may be of interest to quote them at some length.

In the '*Rivista Clinica di Bologna*' for May, 1874, Dr. Ettore Marchiafava of Rome records a case of primary cancer of the lung, of the cylindrical variety, with secondary deposits in the brain and frontal bone. The patient was a mason, æt. 40, who had been under his care in August, 1871. He had been subjected to great and rapid variations of temperature, in consequence of which (as he supposed) he was taken ill. Increasing malaise from day to day was followed by cough, of a convulsive nature, which became more and more frequent and hard. He lost flesh.

In January, 1873, he suffered from attacks of neuralgia in his loins and thighs, and he was then admitted into the San Spirito Hospital, where his disease was diagnosed as chronic tubercular pneumonia.

In February he commenced to complain of supra-orbital pain, which came on in fits and starts, and he occasionally wandered and was never entirely himself. He had an unquenchable thirst. These symptoms, added to those already described, led to the diagnosis of secondary tubercles in the meninges of the brain.

He died on March 19th.

At the autopsy, the meninges were found to be perfectly healthy, but on the base of the brain there were several nodules of new formation. On cutting into the brain similar nodules were seen scattered throughout the cerebral substance, varying in size from a millet seed to a nut. They had a rounded form, were composed of a greyish, soft, granular material, pigmented in places, and could be completely and easily enucleated from the nervous substance. A few similar growths were found in the cerebellum; the pons and medulla were not invaded.

The abdominal organs were all healthy.

In the thorax the pleura, pericardium, and heart were normal.

Scattered throughout the substance of the lungs were nodules similar to those found in the brain, some of them undergoing degeneration, and leading in this way to the formation of small cavities.

The microscopical examination showed an alveolar structure, which was filled with cellular elements having the characters of cylindrical epithelium. In the interior of the alveoli the peripheral cells were arranged along the walls, and presented the genuine form of cylindrical epithelium. In the areas of the alveoli bordering the cancerous nodules was a quantity of catarrhal exudation. The blood-vessels were distended with blood.

The tumours in the brain presented the same sort of fundamental structure. Besides the cylindrical cells the alveoli of the cerebral tumours contained granules of hæmatoidin, indications of old interstitial hæmorrhages. There were evidences of an abundant proliferation of nervous elements in the neighbourhood of the cancerous mass.

The tumours in the frontal bone springing from the periosteum had the same histological type as those in the lungs and brain.

There are no drawings of the microscopical appearances in this case.

The other case, by M. Lataste, Interne of the Paris hospitals, is recorded in the 'Bulletin' for 1875, of the Anatomical Society of Paris. The following is an abstract of the case.

M. F—, æt. 47, a milliner, was taken into the Hôpital Cochin on 11th October, 1875, under M. Bucquoy. The patient had always enjoyed good health, and had the appearance of having a strong constitution. A month before she was first seen she complained for the first time of giddiness and palpitations. Her condition shortly became worse, and she was seen again by her doctor who detected symptoms of left pleuritic effusion. Dyspnœa came on and got worse from day to day, while at the

same time sub-crepitant râles appeared on the other side. There was but slight expectoration, no traces of blood. A week later the dyspnœa became extreme and a condition of semi-coma supervened. There was a little dry cough, which came on in paroxysms, and which was accompanied by a very slight mucous expectoration. Examination of the left side showed absolute dulness reaching as high as the spine of the scapula. On the right side there was some dulness in front, with increased vocal fremitus. Tactile fremitus was completely absent on the left side, but was increased on the right. The heart was slightly displaced towards the right side, but there was nothing abnormal about its sounds. Temperature was 101·5°. The patient died, and at the autopsy the lungs were found studded over with tumours of sizes varying from a pea to a cherry. The interior of some of the larger tumours was yellowish and occasionally pigmented. The lymphatic glands and the pleuræ were also affected; the other organs all healthy.

The microscopic examination of this new growth is recorded and figured by M. Melassez in the 'Archives de Physiologie' for 1876, p. 353. We may briefly state that the histological characters and the description given correspond very nearly with our own.

M. Melassez states in his paper that he has seen one other similar case, for the notes of which he was indebted to Dr. Renaut, but the case is very briefly given and is not illustrated.

We have called this a case of primary cylindrical epithelioma of the lung, with secondary deposits in the pleura, bronchial glands, and liver. That the disease was primary in the lung there can be no reasonable doubt:—*First*, from the wide extent of the pulmonary growth, the whole of both lungs being studded throughout, while there were but three small nodules in the liver, which in this case was the only other organ where the disease could possibly have originated. *Second*, the histological

examination bears out this supposition, the large size and orderly arrangement of the constituent cells in the lung contrasting strikingly with the bastard arrangement in the liver. *Third*, that the lung is just as likely to be the starting-point of such a growth may be inferred from the history of the development of this organ, composed as it is of derivatives from hypoblastic as well as mesoblastic elements of the ovum. It will further be remembered that the bronchial tubes, almost to their terminations, are lined by columnar (ciliated) epithelium, and though this is replaced in the alveoli by an epithelium, the cells of which, owing probably to mechanical causes, have assumed a more flattened type, it is not difficult to imagine that they would serve as a very suitable nidus for the development of true cylindrical epithelioma.

We would take this opportunity of tendering our acknowledgments to Mr. Alfred G. Williams, the house-physician at the Royal Hospital for Diseases of the Chest, for many notes in connection with the case.

DESCRIPTION OF PLATES XV AND XVI.

Primary Cylindrical Epithelioma of the Lung.

(D. W. Finlay, M.D., and R. W. Parker.)

PLATE XV.

Fig. 1. Appearance of the cancer within the alveoli of the lung. The alveolar walls not invaded. As seen by a low power. $\times 125$.

Fig. 2. Appearance of the cancer within the alveoli, but separated from the rest of the lung-substance by a thick wall. The new growth has, in fact, taken the place of the lung-tissue. As seen by a low power. $\times 125$.

Fig. 3. Showing the new growth within the alveoli; the septa perfectly free. The cylindrical character of the epithelial cells is well seen, as also their mode of arrangement. $\times 350$.

Fig. 4. A bronchial gland, showing the cylindrical tubes lined with epithelium. There was some caseous matter in the immediate neighbourhood of this section. $\times 350$.

PLATE XVI.

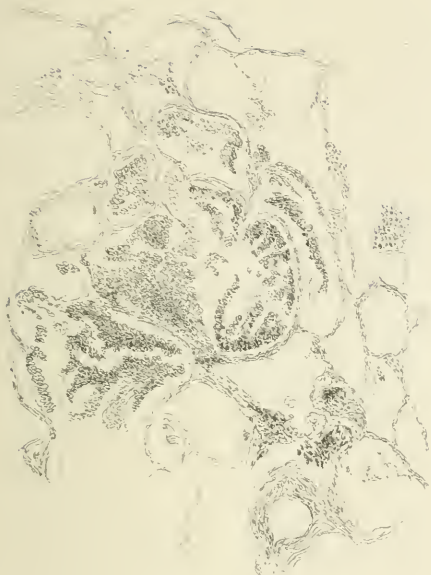
Fig. 5. A nodule from the pleura. There is the same tendency of the epithelial cells to be aggregated in cylinders. Some are larger than others; the large ones consist of a coalescence of smaller ones. $\times 125$.

A refers to the same part as A in Fig. 6, where it is more highly magnified.

Fig. 6. A portion of Fig. 5 (A) more highly magnified, showing the cylindrical nature of the cells and mode of arrangement. $\times 350$.

Fig. 7. From the liver. The cylindrical arrangement of the cells is less typical, though their outline is still characteristic. $\times 350$.

FIG. 1



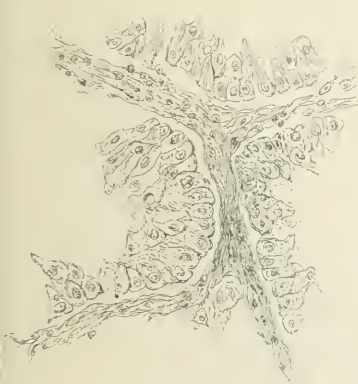
X 125

FIG. 2



X 125

FIG. 3



X 350

FIG. 4



X 250

FIG. 5.



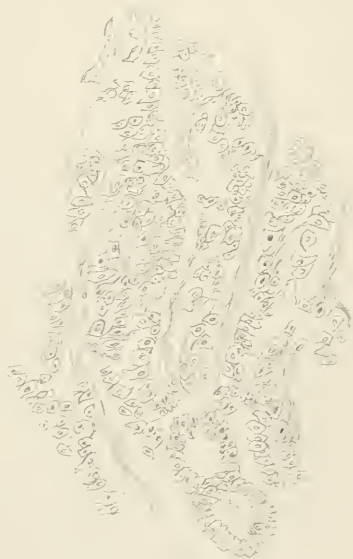
x 125.

FIG. 6.



x 350.

FIG. 7.



x 250.

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